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Exposure to Hazardous Air Pollutants in Homes

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Dedication

To Dr. John Ledbetter

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Exposure to Hazardous Air Pollutants in Homes

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Prior studies have found that human exposure to hazardous air pollutants (HAPs) occurs in homes; however, the depth of these assessments was limited by the extent of the analyzed data. The present Ph.D. dissertation focused on air contaminants of concern in residential buildings, the possible sources of these pollutants, and population subgroups with greater contaminant risk. This research also evaluated the effects of building characteristics and household activity patterns on indoor pollution and risk levels. To this end, an in-depth analysis was performed of data from the Relationships of Indoor, Outdoor and Personal Air (RIOPA) study, one of the most comprehensive exposure assessments to date.

Using personal concentrations from the RIOPA study, a cancer risk assessment was performed to identify both important pollutants and populations at higher risk. The analyzed compounds were acetaldehyde, benzene, chloroform, carbon tetrachloride, *p*-dichlorobenzene (*p*-DCB), ethylbenzene, formaldehyde, methylene chloride, methyl *tert*-butyl ether (MTBE), styrene, trichloroethylene and tetrachloroethylene. Results indicate

that Hispanics and non-Hispanic whites had median cumulative cancer risks (*CCR*) of 520×10^{-6} and 440×10^{-6}, respectively, for which the main contributors were formaldehyde, *p*-DCB, acetaldehyde, chloroform and benzene. Statistically significant differences in *CCR* between and within Hispanic and whites were primarily due to exposures to *p*-DCB. Exposure to formaldehyde was further investigated because this compound was the largest contributor to *CCR* for 69% of Hispanics and 88% of whites, and because most participants had similar cancer risks from these exposures (median = 260×10^{-6}, coefficient of variance = 28%). Results suggest that the U.S. population may be experiencing chronic exposures because of long-term formaldehyde emissions from pressed-wood materials bound with urea-formaldehyde resins. Source removal may be the most effective way to decrease these chronic exposures. Benzene was also examined further because it is a known human carcinogen. Results show that indoor benzene concentrations increased as the proximity of parked vehicles decreased. Residing in a home with an attached garage could lead to exposures to benzene ten times higher than while commuting in a car in heavy traffic, and with mean excess cancers of 17×10^{-6}. Detached garages could reduce health risks from exposure to benzene and other gasoline-related pollutants.

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1. INTRODUCTION

1.1. PROBLEM STATEMENT

Chronic and acute human exposure to hazardous air pollutants occurs in homes. Numerous inhalation exposure studies conclude that personal concentrations of air contaminants, which are monitored in the breathing zone, are typically higher than outdoor concentrations (Adgate et al. 2004; Clayton et al. 1999; Payne-Sturges et al. 2004; Sax et al. 2004; Wallace et al. 1987). The U.S. Environmental Protection Agency (EPA) has classified many of these compounds as hazardous air pollutants (HAPs). Exposure to these HAPs appears to be heavily influenced by indoor pollution in residences because on average Americans spend approximately 70% of their time in their homes (Klepeis et al. 2001), and because personal concentrations are typically higher or comparable to residential indoor concentrations (Adgate et al. 2004; Clayton et al. 1999; Payne-Sturges et al. 2004; Sax et al. 2004; Wallace et al. 1987). Although results from these and other exposure studies provide compelling evidence of the importance of HAPs generated within homes, limitations of these assessments include a lack of information on building characteristics and/or household activity patterns that can considerably affect exposure; a small number of sampled HAPs; or the evaluation of a very specific population subgroup.

This dissertation involved a comprehensive analysis of data from the Relationships of Indoor, Outdoor and Personal Air (RIOPA) study, a relatively recent exposure study in the U.S. that addressed many of the information gaps of previous assessments. Data from the RIOPA study were used to identify air contaminants of concern, their possible sources, and population subgroups with greater contaminant risk; and to evaluate the effect of building characteristics and household activity patterns on

indoor pollution and risk levels. Results from this evaluation were used to recommend strategies to reduce exposure to HAPs.

1.2. OBJECTIVES

The present research involved an evaluation of human exposure to hazardous air pollutants in homes by examining associations among (1) personal, indoor, and outdoor concentrations; (2) building characteristics; (3) demographic factors; and (4) household activity patterns. The objectives were to:

1. Identify air contaminants of concern and their possible sources
2. Identify population subgroups at higher risk from exposure to certain HAPs
3. Evaluate the effects of building characteristics and household activity patterns on exposure to air pollutants
4. Examine possible strategies to reduce exposure to HAPs

1.3. SCOPE

This dissertation is based on the analysis of data from the RIOPA study. From 1999 to 2001, the RIOPA study involved the recruitment of non-smoking adults who resided in Los Angeles County, California, Elizabeth, New Jersey, and Houston, Texas. Approximately 100 adults volunteered in each city; participants in Houston and Elizabeth constitute a sample of convenience, while the Los Angeles participants came from a randomly selected sample of individuals. About 65% of the homes were located close to major outdoor sources of pollution.

The RIOPA database consists of information highly relevant to the assessment of human exposure to air contaminants in homes. The measurements include concurrent personal, indoor, and outdoor concentrations of 16 volatile organic compounds (VOCs) and 10 carbonyls; these compounds commonly originate from indoor and/or outdoor

sources and have been categorized by the EPA as HAPs. Building characteristics such as whole-house air exchange rate (AER) and indoor temperature were simultaneously measured during the RIOPA study. Demographic measures, collected with questionnaires, include ethnicity, income and gender. Some of the household activity patterns recorded during the monitoring period include the opening of windows for ventilation, the use of air fresheners, and the location of parked vehicles.

The RIOPA database was analyzed in three phases:

- **Phase 1:** Assess cancer risks from exposure to HAPs
- **Phase 2:** Evaluate long-term formaldehyde concentrations in homes
- **Phase 3:** Examine the effects of parked cars on indoor BTEX and MTBE concentrations

In the first phase, a cancer risk assessment from exposure to HAPs was performed to address objectives (1) and (2), and to a lesser extent objectives (3) and (4). Results from Phase 1 led to formaldehyde and benzene as pollutants of concern that required further investigation. Thus, formaldehyde and benzene were evaluated in the second and third phases, respectively, to focus on objectives (3) and (4) in more depth. Benzene, toluene, ethylbenzene, xylenes (BTEX) and MTBE were also examined in Phase 3 because these compounds were also emitted by gasoline vapors and car exhaust in Los Angeles, Elizabeth and Houston during the RIOPA study.

1.4. ORGANIZATION

This dissertation is divided into two major parts. The first part is an executive summary that includes a literature review, an overview of the methodology, and a summary of the results. This material generally follows the three research phases presented in Section 1.3. The second part consists of Appendices A, B and C, each of which respectively includes the complete text for the supporting papers that describe in

detail the three research phases. These papers are referenced throughout this dissertation and are listed below:

- **Appendix A:** Hun, D. E., Siegel, J. A., Morandi, M. T., Stock, T. H., Corsi, R. L. 2009. Cancer risk disparities between Hispanic and non-Hispanic white populations: The role of exposure to indoor air pollution. Environmental Health Perspectives 117(12):1925-1931.
- **Appendix B:** Hun, D. E., Corsi, R. L., Morandi, M. T., Siegel, J. A. Formaldehyde in residences: Long-term indoor concentrations and influencing factors. Indoor Air (Accepted to *Indoor Air*).
- **Appendix C:** Hun, D. E., Corsi, R. L., Morandi, M. T., Siegel, J. A. Indoor residential concentrations of BTEX and MTBE (Submitted to *Building and Environment*).

2. BACKGROUND

2.1. INHALATION EXPOSURE ASSESSMENTS

Inhalation exposure assessments have significantly improved the ability to evaluate human exposure to contaminants that are classified by the EPA as hazardous air pollutants. These assessments involve the sampling of personal concentrations in the breathing zone of individuals throughout their daily activities. Consequently, this type of monitoring serves as a more accurate indicator of exposure than ambient concentrations because it takes into account the large fraction of time people spend indoors, the contribution from indoor sources of HAPs, and the penetration of outdoor pollutants into buildings. Exposure assessments usually focus on residences because Americans spend on average nearly 70% of their time in their homes (Klepeis et al. 2001). Therefore, for individuals who do not directly interact with air contaminants at work, exposure to HAPs usually may be dominated by sources present in their homes. Thus, exposure studies usually concurrently measure indoor and outdoor concentrations of HAPs in residences to examine their associations with personal concentrations that may help to identify sources.

The Total Exposure Assessment Methodology (TEAM; Wallace et al. 1987) study was the first major exposure assessment undertaken in the United States and involved human subjects in eight urban areas in the 1980s. An important finding was that for 10 of the 20 sampled VOCs, personal concentrations were sometimes 10 times higher than outdoor values mostly due to sources found indoors such as tobacco smoke, chlorinated tap water and deodorizers/air fresheners (Wallace et al. 1987; Wallace 1991). Approximately a decade later, the National Human Exposure Assessment Survey (NHEXAS; Gordon et al. 1999) reaffirmed the importance of indoor residential sources to exposure to HAPs. More recent investigations include: the School Health Initiative:

Environment, Learning, and Disease (SHIELD; Adgate et al. 2004) study; an assessment in South Baltimore (Payne-Sturges et al. 2004); and the Toxics Exposure Assessment Columbia-Harvard (TEACH; Sax et al. 2004) study. Findings from these investigations show that personal concentrations for formaldehyde, a probable human carcinogen (U.S. EPA 2005), are dominated by indoor sources. This domination was also observed frequently for benzene, a known human carcinogen (U.S. EPA 2005), even though participants were nonsmokers and tobacco smoke is the leading source of benzene for both personal and indoor air concentrations in the general population (Wallace 1996). Findings from these latter investigations supplemented those from TEAM and NHEXAS, given that these more recent studies evaluated exposure to carbonyls, and/or excluded smokers because tobacco smoke contains a large number of air contaminants (Nazaroff and Singer 2003) that may diminish the importance of other sources in nonsmoking homes.

In addition to providing compelling evidence of the importance of indoor pollution, these studies also suggest that certain population subgroups may be experiencing higher exposures to these contaminants. Pellizzari et al. (1999) examined data from NHEXAS and determined that minorities had higher personal measurements for lead and benzene than non-minorities. D'Souza et al. (2009) evaluated air pollutant concentrations from the 1999-2000 National Health and Nutrition Examination Survey (NHANES) and concluded that Hispanics and African-Americans had much higher personal concentrations for BTEX (benzene, toluene, ethylbenzene and xylenes), MTBE and *p*-DCB than whites. Nevertheless, neither of these studies collected information on residential building characteristics that have been shown to affect indoor concentrations of contaminants, such as air exchange rates (Gilbert et al. 2006; Johnson et al. 2004; Sax et al. 2004) and indoor temperature (Matthews et al. 1984; Myers 1985). Furthermore,

large national assessments, such as NHEXAS and NHANES, tend to focus on a smaller number of HAPs and often do not include carbonyls such as formaldehyde. Conversely, studies that collected more extensive information on building characteristics and sampled a much larger number of HAPs, such as TEACH, usually concentrate on specific groups (e.g., high school students), which limits the ability to draw generalizations for the entire population.

2.2. CANCER RISK ASSESSMENTS

Cancer risk assessments have been used to help identify air pollutants of concern. These assessments utilize cancer potency factors that are derived from occupational and animal studies that involve exposure to high doses of a particular air contaminant. Cancer potency factors are extrapolated from these studies to predict upper-bound excess cancers in a population of a million due to exposure to 1 $\mu g/m^3$ of a pollutant throughout a 70-year lifetime. Cancer risks from individual pollutants are added to estimate a cumulative cancer risk (*CCR*; Caldwell et al. 1998). Although cancer risk assessments have limitations due to the methods and assumptions used to derive the cancer potency factors, and due to the manner in which the cumulative effect of various contaminants is estimated, this type of evaluation is a useful tool for exploring risks in a standardized manner.

Numerous researchers have used cancer risk assessments to prioritize the reduction of outdoor contaminants and to identify population subgroups that may be at higher risk from exposure to these pollutants. Outdoor concentrations are acquired from dispersion models or from actual measurements. The Assessment System for Population Exposure Nationwide (ASPEN) dispersion model estimates ambient concentrations at the census tract level by incorporating emissions from several sources (e.g., Toxic Release Inventory (TRI) and estimates for mobile and area sources), atmospheric decay, and

meteorological information (e.g., wind speed and direction). The National Air Toxics Assessment (NATA) combines outdoor concentrations from ASPEN with an inhalation exposure model to predict cancer risks at the census tract level. The South Coast Air Quality Management District (SCAQMD 2008) measured outdoor concentrations that were used in the Multiple Air Toxics Exposure Study III (MATES) to evaluate outdoor contamination in California's South Coast Basin. Woodruff et al. (2000) reported a median *CCR* of 180×10^{-6} in the U.S. after analyzing 1990 ambient concentrations from ASPEN. Morello-Frosch and Jesdale (2006) utilized concentrations of HAPs from the 1996 NATA and estimated a mean *CCR* of 632×10^{-6} for metropolitan areas. The MATES assessment reported a mean *CCR* of $1{,}200\times10^{-6}$ in California's South Coast Basin. These three investigations identified HAPs that are emitted by mobile sources – including diesel particulate matter, benzene and 1,3-butadiene – as contributors that made up at least 65% of the cumulative risk. However, these models do not take into account the contributions of indoor sources to overall HAP exposure.

Two recent studies involved cancer risk assessments using personal concentration measurements. Payne-Sturges et al. (2004) monitored homes (n = 31) in South Baltimore communities that were in close proximity to a high density of chemical industries. They sampled personal, indoor, and outdoor concentrations for nine VOCs of indoor and outdoor origin, and estimated cancer risks using the personal concentrations. The mean *CCR* (183×10^{-6}) was dominated by chloroform (31%), and exposures to this compound were likely influenced by indoor sources because the mean indoor concentration was about seven times higher than the mean outdoor value. The TEACH study involved the measurement of personal concentrations of 11 VOCs and two carbonyls for teenagers in New York City (n = 40) and Los Angeles (n = 41). Exposures to formaldehyde and *p*-dichlorobenzene accounted for at least 86% of the mean *CCR*s for New York (957×10^{-6})

and Los Angeles (806×10^{-6}) (Sax et al. 2006). Personal concentrations for both of these pollutants were probably affected by indoor sources because mean indoor concentrations were between five to 18 times higher than outdoor concentrations. These results and those from Morello-Frosch and Jesdale (2006) and from Woodruff et al. (2000) suggest that different HAPs would be targeted as the pollutants of concern depending on whether the cancer risk assessment is based on personal or outdoor concentrations. Relative to past studies, this dissertation involved a risk assessment with personal concentrations from a larger and more diversified sample group. Furthermore, the present work concurrently evaluated the effects of building characteristics and household activity patterns on exposure to HAPs to identify strategies to reduce risk.

Cancer risk assessments have also been used to determine if population subgroups are disproportionately affected by HAPs. Apelberg et al. (2005) and Morello-Frosch and Jesdale (2006) analyzed data from NATA and concluded that census tracts with larger proportions of Hispanics or African-Americans appear to have higher cancer risk levels than tracts with greater proportions of non-Hispanic whites. However, this evidence is mostly based on outdoor measurements; therefore, these studies identified HAPs emitted by mobile sources as the main contributors to *CCR*. This dissertation expanded the existing knowledgebase on the exposure of population subgroups to air contaminants by using personal concentrations that incorporate the contribution of indoor pollution to possible discrepancies in risk.

3. METHODS

3.1. THE RIOPA STUDY

Data from the RIOPA study were made available by the Health Effects Institute (HEI 2008). From 1999 to 2001, the RIOPA study involved the recruitment of non-smoking adults who resided in Los Angeles County, California, Elizabeth, New Jersey, and Houston, Texas. Participants in Houston and Elizabeth constitute a sample of convenience, while the Los Angeles participants were a subset from a randomly selected sample of individuals from another study. Approximately 100 adults volunteered in each city; most worked at home or their workplace was in the same neighborhood as their residences. About 65% of the homes were located in close proximity to major outdoor sources of pollution such as highways in Los Angeles, petrochemical facilities in Houston, and small sources (e.g., dry cleaners) in Elizabeth.

Participants and their homes were monitored during two 48-hour periods that were approximately three months apart. Air contaminants were selected to include HAPs that are categorized by the EPA as urban air toxics or mobile-source pollutants, as well as compounds from primarily indoor origin. Air samples were collected concurrently in the personal or breathing zone, and inside and outside the house. Sixteen VOCs were monitored using Organic Vapor Monitors (OVM 3500, 3M Company, St. Paul, MN, USA), and ten carbonyls were measured using the Passive Aldehydes and Ketones Sampler (PAKS) coupled with HPLC-fluorescence analysis (Zhang et al. 2001). Concentrations at or below the respective method detection limit (MDL) were censored by replacement with ½ of the MDL concentrations. Demographic and building characteristics, as well as daily indoor and outdoor activity patterns were collected during each of the sampling sessions with questionnaires and walkthrough surveys. Whole-

house air exchange rates (AER) were measured using a perfluorocarbon tracer (PFT) method (Dietz et al. 1986). Temperature and relative humidity were recorded every five minutes using a HOBO sensor (HOBO, Onset Computer Corp, Bourne, MA). Temperature, relative humidity and AER were reported as time-averaged values for the sampling period.

3.2. DATA ANALYSIS

Several conventions were followed throughout this analysis of the RIOPA data. Measurements from a household where someone smoked during a sampling period were excluded. In most instances, demographic data and building characteristics (e.g., building age) from the first visit were selected when information from the first and second sessions were not in agreement. In the case of income, the midpoints of disparate income ranges were averaged. Ventilation rates greater than 5 h^{-1} were also excluded because the PFT method is unreliable at these values. Residences where volumes were recorded to be less than 80 m^3 were not included in the analysis because it is highly probable that these values were not correct.

3.2.1. Cancer Risk Assessment

Cancer risks were used to evaluate the relative importance of sampled pollutants. This investigation focused on 12 of the sampled HAPs for which estimates of cancer unit risk factors are available as shown in Table 1. Risk factors were primarily obtained from the U.S. EPA (2005); however, estimates from the California EPA (CalEPA 2002, 2005) and Caldwell et al. (1998) were used when not available from the U.S. EPA. Only houses with personal concentrations for all of these 12 compounds in either of the monitoring sessions were considered, reducing the overall sample size from 311 to 243. Air concentrations for each pollutant and air exchange rates were averaged when the

household was monitored twice because measurements from the same house are not independent. An estimate of cancer risk associated with each HAP was derived as:

$$CR = C_{per} \times UR \quad (1)$$

where *CR* is the cancer risk, C_{per} is the measured personal concentration (μg/m^3), and *UR* is the inhalation cancer unit risk factor and represents the probability of cancer in a population of a million due to a 70-year exposure to 1 μg/m^3. The cumulative cancer risk (*CCR*) was calculated by summing the cancer risks from all 12 HAPs (Caldwell et al. 1998).

Table 1. Measured hazardous air pollutants in RIOPA with available cancer unit risk factors.

Compound	WOE		Unit risk	Source[c]
	IRIS[a]	IARC[b]	(m^3/μg)	
Acetaldehyde	B2	2B	2.2×10^{-6}	1
Benzene	A	1	7.8×10^{-6}	1
Carbon tetrachloride	B2	2B	1.5×10^{-5}	1
Chloroform	B2	2B	2.3×10^{-5}	1
Ethylbenzene	NC	2B	2.5×10^{-6}	2
Formaldehyde	B1	1	1.3×10^{-5}	1
Methylene chloride	B2	2B	4.7×10^{-7}	1
MTBE	NC	3	2.6×10^{-7}	3
p-Dichlorobenzene	NC	2B	1.1×10^{-5}	3
Styrene	NC	2B	5.0×10^{-7}	4
Trichloroethylene	NC	2A	2.0×10^{-6}	3
Tetrachloroethylene	NC	2A	5.9×10^{-6}	3

Abbreviations: IARC, International Agency for Research on Cancer; IRIS, Integrated Risk Information System; MTBE, methyl *tert*-butyl ether; WOE, weight of evidence.
[a]IRIS classification: A, known carcinogen; B1, probable carcinogen; B2, probable carcinogen; NC, not classified.
[b]IARC classification: 1, carcinogenic; 2A, probably carcinogenic; 2B, possibly carcinogenic; 3, not classifiable as to carcinogenicity to humans.
[c]Sources: 1, IRIS (U.S. EPA 2005); 2, CalEPA (2002); 3, CalEPA (2005); 4, Caldwell et al. (1998).

3.2.2. Long-Term Formaldehyde Concentrations

Only homes with values for indoor and outdoor formaldehyde concentrations, AER, and indoor temperature were used in the analysis. Averages were calculated when all of these measurements were available for the two monitoring sessions because these are dependent variables that describe a single household. These constraints reduced the overall sample size from 311 to 179.

3.2.3. BTEX and MTBE Concentrations from Parked Cars

Apartments were not included in the evaluation because pollutants from adjacent dwellings can infiltrate through shared walls and affect the measured concentrations. Households that had gasoline-powered devices other than vehicles inside the house were also excluded from the assessment. Data from monitoring sessions where information on the location of the parked car was missing were additionally excluded. Only data from homes with values for indoor and outdoor BTEX and MTBE concentrations, and ventilation rates were employed. These constraints reduced the overall sample size from 311 to 114.

A cross-sectional analysis was performed using homes for which information was available for only one of the visits or homes where cars were parked in the same location in both sampling periods. Averages were calculated when indoor and outdoor BTEX and MTBE concentrations as well as AER were available for the two monitoring sessions because these are dependent variables that describe a single household.

3.2.4. Statistical Analysis

Nonparametric statistical analyses were utilized because the data generally had a positively skewed distribution. Associations between variables were evaluated with Spearman rank-correlation coefficients (r_s). The Wilcoxon sign-rank test was used to

assess differences between paired samples, such as concurrent indoor and outdoor concentrations. The Wilcoxon rank-sum test was utilized to evaluate differences between two independent samples, such as indoor concentrations from homes with parked vehicles next to the living area and residences without such sources. Similarly, the Kruskal-Wallis test was used with three or more levels. Differences were considered statistically significant at $p \leq 0.05$. SPSS (version 15.0, SPSS Inc.) was employed for these analyses.

4. RESULTS AND DISCUSSION

4.1. CANCER RISK ASSESSMENT

The cumulative cancer risks (*CCR*s) for all participants in the RIOPA study (n = 238) were greater than 10^{-4}. Mean, median and 90th percentile *CCR*s were $1{,}100\times10^{-6}$, 480×10^{-6} and $1{,}700\times10^{-6}$, respectively, after excluding two unusually high measurements for chloroform (1,224 μg/m^3) and tetrachloroethylene (1,340 μg/m^3). Formaldehyde, *p*-dichlorobenzene (*p*-DCB), acetaldehyde, chloroform and benzene contributed 93% of the mean *CCR*. For individuals with the highest risks (i.e., top 10th percentile), *p*-DCB accounted for 91% of the mean *CCR*.

4.1.1. Differences between Hispanics and non-Hispanic Whites and Air Pollutants of Concern

Hispanics are the largest and the fastest-growing minority group in the United States, and minimal information is available regarding their exposures to air contaminants. As such, this dissertation focused on Hispanics and their risks relative to non-Hispanic whites. Tables A2 and A3, which are Tables 2 and 3 in Appendix A, summarize personal concentrations and household characteristics for both of these population subgroups in Los Angeles, Elizabeth and Houston. Estimated risks based on personal concentrations are shown in Figure 1. Hispanics and whites had cancer risks for nine of the twelve pollutants that were higher than the EPA benchmark of 10^{-6}. Differences in risk between the two ethnic groups varied by city. The *CCR* was higher among Hispanics than whites in Elizabeth ($p \leq 0.05$) and Houston ($p \leq 0.01$) mostly because of differences in exposure to *p*-DCB, and to a lesser extent to benzene and chloroform. The median *CCR* of Hispanics in Elizabeth, 500×10^{-6}, was 1.2 times higher than that for whites. This ratio was 1.6 in Houston, where Hispanics had a median

cumulative risk of 720×10^{-6}. For Los Angeles, the median *CCR* was about 440×10^{-6} for both ethnic groups.

Cumulative risk tertiles were used to identify the compounds that affect individuals with non-extreme and extreme cancer risk levels. The average contribution of each HAP to the mean of *CCR* tertiles is shown in Figure 2. In general, most of the 1st and 2nd tertiles for Hispanics and whites were somewhat similar with mean cumulative values ranging from 280×10^{-6} to 770×10^{-6}. Formaldehyde contributed from 35 to 77% of the mean *CCR*, while acetaldehyde, benzene, chloroform and *p*-DCB accounted for 18 to 36%. Increases in risk from the 2nd to 3rd tertiles were primarily due to *p*-DCB for both ethnic groups: *p*-DCB was responsible for 53 to 88% of the mean *CCR* among Hispanics and for 28 to 64% among whites. Hispanics in Houston had the highest mean *CCR* for the 3rd tertile ($5{,}500\times10^{-6}$), of which 88% was due to *p*-DCB. Other exposure studies have also reported high personal *p*-DCB concentrations among Hispanics (Adgate et al. 2004; Churchill et al. 2001; D'Souza et al. 2009; Sax et al. 2006). Formaldehyde was the second most important pollutant for all of the 3rd tertiles, contributing 7 to 34% of the mean *CCR* among Hispanics and 10 to 60% among whites. The *CCR*s for Hispanics and whites in the top two tertiles remained statistically different (i.e., Hispanics > whites) after *p*-DCB was excluded from the cumulative risk calculations in Elizabeth and Houston, mainly because of differences in exposure to benzene and chloroform. However, actual differences in *CCR* were greatly diminished following the removal of *p*-DCB.

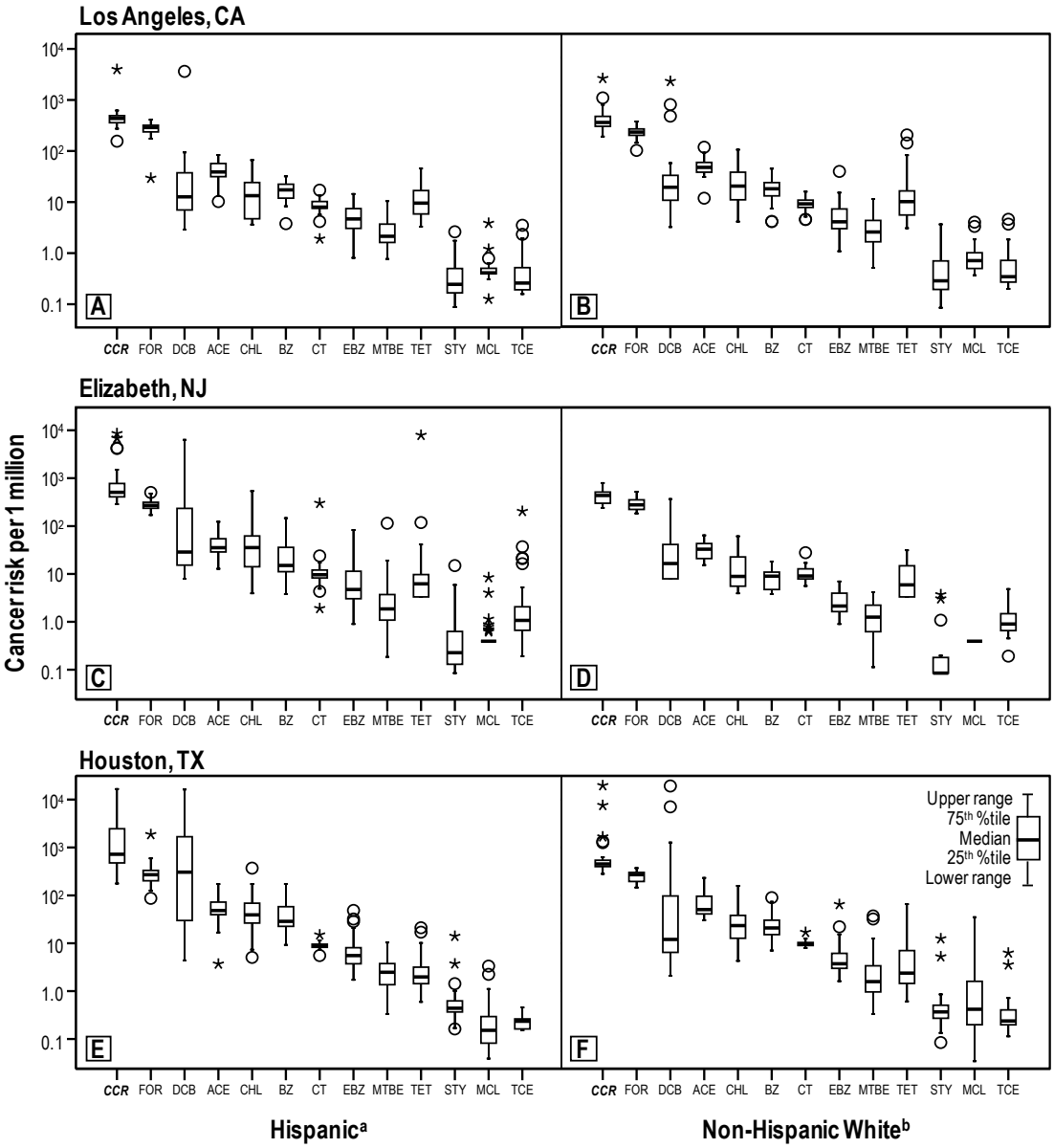

Figure 1. Distributions of cancer risks based of Hispanics and whites in Los Angeles (*A* and *B*), Elizabeth (*C* and *D*), and Houston (*E* and *F*).

'o' and '*' indicate values between 1.5 and 3, and > 3 times the box length, respectively, from the 25th or 75th percentiles. Hispanic: Los Angeles, n = 23; Elizabeth, n = 54; Houston, n = 44. Non-Hispanic white: Los Angeles, n = 43; Elizabeth, n = 15; Houston, n = 36. Abbreviations: ACE, acetaldehyde; BZ, benzene; CCR, cumulative cancer risk; CHL, chloroform; CT, carbon tetrachloride; DCB, *p*-dichlorobenzene; EBZ, ethylbenzene; FOR, formaldehyde; MCL, methylene chloride; MTBE, methyl tert-butyl ether; STY, styrene; TCE, trichloroethylene; TET, tetrachloroethylene.

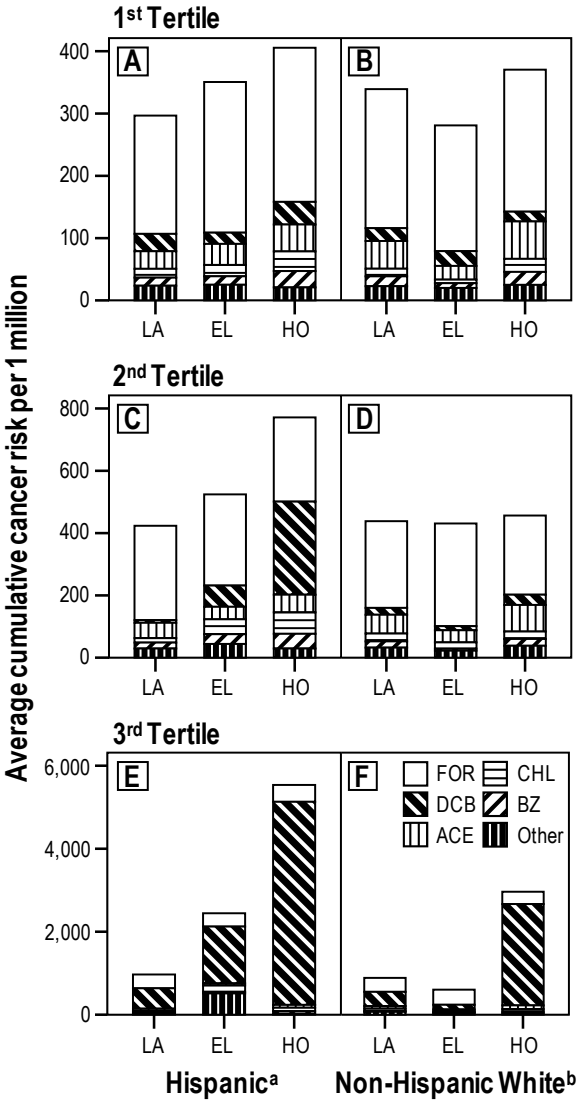

Figure 2. Average of 1st (*A* and *B*), 2nd (*C* and *D*), and 3rd (*E* and *F*) *CCR* tertiles for Hispanics (*A*, *C* and *E*) and non-Hispanic whites (*B*, *D* and *F*).

Every tertile shows the average contribution of each HAP. Hispanic: LA: $n = 23$, EL: $n = 54$, HO: $n = 44$. Non-Hispanic white: LA: $n = 43$, EL: $n = 15$, HO: $n = 36$. Abbreviations: ACE, acetaldehyde; BZ, benzene; CHL, chloroform; DCB, *p*-dichlorobenzene; EL, Elizabeth; FOR, formaldehyde; HO, Houston; LA, Los Angeles; Other, carbon tetrachloride, ethylbenzene, methylene chloride, methyl *tert*-butyl ether, styrene, tetrachloroethylene, trichloroethylene.

Among the few studies that have examined cancer risks using personal concentrations, the Toxics Exposure Assessment Columbia-Harvard study (TEACH; Sax et al. 2006) was selected for comparison because, except for 1,3-butadiene, both RIOPA and TEACH considered the same compounds and unit risk factors. The TEACH study involved mostly participants from minority backgrounds in New York City (African-American = 43%, Hispanic = 50%) and Los Angeles (Hispanic = 93%), although it only included high school students. The risk estimates from TEACH and RIOPA share similarities such as *CCR* values (TEACH: New York median = 670×10^{-6}, Los Angeles median = 490×10^{-6}), compounds that were identified as the main contributors to *CCR*, cancer risks from exposure to formaldehyde (TEACH median = 240×10^{-6}), and *p*-DCB as the HAP responsible for the largest discrepancies in risk within the study.

4.1.2. Sources

The possible origin of individual HAPs were explored by examining statistical associations between personal (C_{per}) and indoor (C_{in}), and personal and outdoor (C_{out}) concentrations. Results from this evaluation are included in Table A2. In general, the analyses for both ethnic groups indicate that C_{per} and C_{in} were similar, and that C_{per} was higher than C_{out} ($p \leq 0.01$). For the majority of the studied compounds, personal and indoor concentrations were probably influenced by the same source(s), and most of the exposure occurred indoors. In a few cases, C_{per} was statistically higher than C_{in}, implying short episodic events during which the participant may have been close to sources. Among the five compounds that were the main contributors to *CCR* (i.e., acetaldehyde, benzene, chloroform, formaldehyde and *p*-DCB), exposure to all of these pollutants but benzene appear to have been dominated by indoor residential sources. The discussion that follows focuses on these five compounds.

The disproportionate effect from *p*-DCB may be caused by the higher frequency at which certain consumer products appear in Hispanic than in white households. Typical indoor sources of *p*-DCB include deodorizers/air fresheners and moth repellents (Wallace 1991), which are often pure *p*-dichlorobenzene and are prone to relatively high mass emission rates. Answers to RIOPA questionnaires suggest that deodorizers/air fresheners are more prevalent among Hispanics than are moth repellents; 59% reported to have used air fresheners during the study, while only 6% utilized moth repellents. The TEACH study concluded that in the Los Angeles households, most of which were Hispanic, 75% used air fresheners and less than 20% used moth repellents (Sax et al. 2004). Air fresheners containing *p*-DCB may be more frequently used in low-income Hispanic homes given that data from RIOPA indicates that households with annual incomes below $25,000 had median risks from exposure to *p*-DCB that were four times higher than those estimated in homes with higher family earnings ($p \leq 0.01$). Among the wide variety of air fresheners, Churchill et al. (2001) and Serrano-Trespalacios et al. (2004) noted that solid toilet bowl deodorants appear to be commonly used by Mexican Americans.

Formaldehyde was the largest contributor to *CCR* for 69% of Hispanics and 88% of whites. The TEACH study also concluded that formaldehyde was generally the highest contributor to *CCR* among participants with non-extreme risk values (Sax et al. 2006). Although formaldehyde is ubiquitous in the outdoor air of urban areas as a byproduct of fuel combustion and photochemistry, indoor sources tend to dominate human exposure. Numerous products, such as permanent-press fabrics, paint and nail polish remover, emit this compound (Gupta et al. 1982; Kelly et al. 1999). However, pressed-wood materials bound with urea-formaldehyde resins are likely the main indoor source in most homes given that these products are widespread in the form of cabinetry,

furniture and building materials (Garrett et al. 1997; Gupta et al. 1982; Kelly et al. 1999; Mølhave et al. 1999).

In the homes of nonsmokers, outdoor sources may be of more importance with respect to exposures to benzene than are indoor sources (Wallace 1996). This was observed in Los Angeles where personal and indoor benzene concentrations were statistically similar to outdoor values for both Hispanics and whites. However, C_{per} and C_{in} were statistically greater than C_{out} in Elizabeth and Houston even though 42% and 92% of these homes were located less than 1 km from major ambient sources of HAPs (e.g., petrochemical facilities), respectively. Furthermore, C_{per} and C_{in} were statistically similar in these homes. These results suggest that personal and indoor concentrations were probably affected by the same source(s), and that this source was not entirely of ambient origin. Emissions from gasoline-powered devices were likely a major contributor to exposure given that air contaminants from attached garages tend to infiltrate into the occupied space of homes (Batterman et al. 2007; Dodson et al. 2008; Thomas et al. 1993).

The main residential source of chloroform is volatilization from chlorinated tap water, which has chloroform concentrations that are highly variable depending on the water source and date. Higher personal concentrations of chloroform among Hispanics may be because these households tend to exceed the U.S. average number of people per home by a factor of 1.35 (U.S. Census Bureau 2004), which may lead to a larger than average number of showers and other water uses, from which chloroform can volatilize, per residence. Nuckols et al. (2005) determined that chloroform concentrations in the blood and breath are affected by emissions that occur while others are taking showers. Among Hispanics, women (upper 50^{th} percentile risk range = 36×10^{-6} to 540×10^{-6}) had statistically higher risks than men (upper 50^{th} percentile risk range = 13×10^{-6} to 140×10^{-6})

from exposures to chloroform ($p \leq 0.01$) probably because household activities that involve the use of chlorinated tap water and that increase contact with this pollutant, such as washing dishes (Nuckols et al. 2005), are more often performed by women.

Acetaldehyde was among the important contributors to cumulative cancer risk for both Hispanics and whites. Exposures to acetaldehyde were of significance to *CCR* in the TEACH study (Sax et al. 2006), and indoor sources appeared to have been dominant over outdoor sources (Sax et al. 2004). Analysis of the RIOPA data indicate statistically significant associations between acetaldehyde and benzene for both personal (r_s: Hispanics = 0.22, whites = 0.32) and indoor (r_s: Hispanics = 0.24, whites = 0.30) concentrations. This suggests that combustion-related sources other than tobacco smoke, which was excluded from the RIOPA study, may have influenced exposures to acetaldehyde. Other possible indoor sources include detergents, cleansers and liquid wax (Nazaroff and Weschler 2004).

4.1.3. Air Exchange Rates

Previous work has indicated that house ventilation rates may be among the most important building characteristics to influence indoor concentrations of pollutants (Gilbert et al. 2005; Johnson et al. 2004; Sax et al. 2004). In general, Hispanics and whites who lived in houses with low ventilation rates had higher cancer risks from exposure to HAPs, particularly from *p*-DCB and chloroform, consistent with results from the TEACH study (Sax et al. 2004). The cumulative effect of AER on exposure was demonstrated by statistically significant differences in *CCR* between participants who lived in homes with ventilation rates below 0.5 h^{-1} and above 1 h^{-1}. Moreover, higher median AERs in Hispanic households in Los Angeles (1.2 h^{-1}) than in Elizabeth (1.0 h^{-1}) and Houston (0.5 h^{-1}) may explain why (1) Hispanics in Los Angeles had lower *CCR*s than in the other two cities, (2) no statistical differences in *CCR* were observed in Los

Angeles between Hispanics and whites (median AER for white households = 0.8 h^{-1}), and (3) personal and outdoor concentrations for benzene, which are mostly generated by mobile sources, were statistically similar for Hispanics in Los Angeles. Differences in AER among cities may be because a larger percentage of Hispanic homes in Los Angeles (74%) reported to have had their windows open for some time during the sampling session than in Elizabeth (30%) and Houston (7%). Even though these results suggest that ventilation rates can reduce risks from HAPs, this measure is not sufficient. People in homes with AERs 2.9 times higher than a recommended value of 0.35 h^{-1} (ASHRAE 2004) experienced median *CCR*s of 440×10^{-6}, a risk that is two orders of magnitude greater than the EPA benchmark of 10^{-6}.

4.1.4. Further Investigations

Exposure to formaldehyde is evaluated in more depth in Section 4.2 for several reasons. First, the EPA has classified formaldehyde as a probable human carcinogen (Group B1). Second, the RIOPA study and other investigations resulted in similar median indoor concentrations of about 20 $\mu g/m^3$ (Gordon et al. 1999; Sax et al. 2006; Weisel et al. 2005), which suggests that the U.S. population may be subjected to chronic exposures that can lead to cancer risks of 260×10^{-6}. Third, information is lacking regarding reasons behind the apparent persistence of formaldehyde concentrations in existing homes and how these are affected by factors that tend to influence formaldehyde concentrations in new homes. Lastly, the literature does not include information on strategies to reduce exposure to formaldehyde in existing homes.

Residential sources of benzene were also studied, and reported further in Section 4.3, for several reasons. First, the EPA has classified benzene as a known human carcinogen (Group A). Second, cancer risk assessments that used either ambient concentrations or personal concentrations from nonsmoking populations have repeatedly

identified benzene as a pollutant of concern (Loh et al. 2007; Payne-Sturges et al. 2004; Sax et al. 2006; SCAQMD 2008; Woodruff et al. 2000). Third, researchers have reported indoor benzene concentrations to be higher than outdoor concentrations in homes with occupants who do not smoke, which indicates the presence of sources such as gasoline-related devices in close proximity to the living area (Batterman et al. 2007; Thomas et al. 1993; Weisel et al. 2005). Fourth, the RIOPA study included measurements of MTBE concentrations, a good tracer for gasoline-related emissions; MTBE was in use in Los Angeles, Elizabeth and Houston during the collection of air samples. Lastly, data from the RIOPA study provided the opportunity to evaluate how the proximity of parked vehicles affected indoor concentrations of gasoline-related pollutants, the origin of these contaminants (i.e., vapor or tailpipe emissions), and the effect of ventilation rates on indoor concentrations for these VOCs.

4.2. LONG-TERM FORMALDEHYDE CONCENTRATIONS

In this part of the dissertation, most of the assessed homes were located in Los Angeles (n = 73), followed by Elizabeth (n = 58) and Houston (n = 48). The majority of these residences were either single-family detached homes (50%) or apartments (37%). Of the 179 homes, 23 were less than 5 years old, of which 21 were apartments in Los Angeles. Ten out of 12 manufactured homes were older than 5 years; building age was not available for one of these houses. Data from Los Angeles, Elizabeth and Houston were combined because of the small samples sizes. Table B1 presents summary statistics for indoor formaldehyde concentrations and other building-related measurements. More than 99% of the formaldehyde concentrations were above the method detection limit of 0.14 to 1.0 ppb. Indoor formaldehyde had mean and median concentrations of approximately 17 ppb, and a relatively small coefficient of variation (CV) of 26%. Personal concentrations had a median value of 17 ppb, which exceeded California's

Reference Exposure Level of 7 ppb (OEHHA 2008). Moreover, C_{per} were statistically similar to C_{in}, but statistically higher than C_{out}, which suggests that exposures were highly related to indoor residential sources

4.2.1. Sources

Indoor concentrations were statistically higher ($p \leq 0.01$) than outdoor concentrations, which indicates that indoor sources were a major contributor to indoor formaldehyde concentrations. The percentage contribution of indoor sources to indoor concentrations (I_{cont}) was calculated by subtracting C_{out} from C_{in} and dividing the result by C_{in}. The cumulative distribution curve for I_{cont} is shown in Figure 3 and indicates that in half of the RIOPA houses more than 70% of C_{in} originated from indoor sources. Additionally, the contribution of indoor sources to C_{in} was greater than that from outdoor sources for 90% of the homes. Figure 3 also shows estimated I_{cont} for new homes based on indoor concentrations published by Sherman and Hodgson (2004) and an outdoor formaldehyde concentration of 2 ppb from a related study by Hodgson et al. (2000). Their investigations involved seven manufactured and seven conventional homes less than one year old. Indoor sources accounted for at least 95% of C_{in} in half of the new homes.

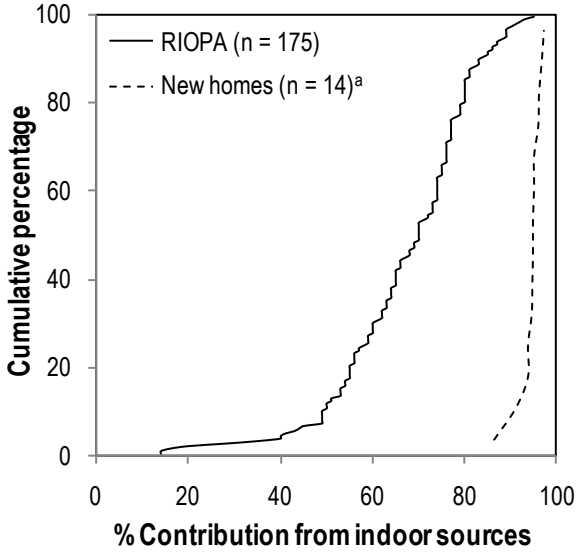

Figure 3. Cumulative distribution functions for the percent contribution from indoor sources to indoor concentration.
[a]Data from Hodgson et al. (2000) and Sherman and Hodgson (2004).

There are numerous indoor sources of formaldehyde, including consumer products such as permanent-press fabrics and fiberglass products (Gupta et al. 1982; Kelly et al. 1999), chemical reactions with ozone (Morrison and Nazaroff 2002; Weschler 2000), and pressed-wood products (Gupta et al. 1982; Kelly et al. 1999). The contribution to C_{in} from chemical reactions involving ozone and unsaturated organic compounds with a terminal carbon-carbon double bond was explored for the Houston homes. Indoor formaldehyde concentrations were estimated with a procedure similar to the one described by Loh et al. (2008). Outdoor ozone concentrations were obtained from the Texas Commission on Environmental Quality database (TCEQ 2009), and were based on the RIOPA sampling date and the monitoring station that was closest to the residence. Results showed no statistical associations between C_{in} and ozone concentrations, but these may have been partly influenced by the use of air conditioning systems in the Houston homes during a significant part of the sampling period (mean =

69%, median = 96%). This would limit ozone infiltration to occur through cracks in the building envelope, which may increase the loss of ozone due to chemical reactions with building envelope materials. Zhang et al. (1994) also reported poor correlations between C_{in} and indoor ozone concentrations due to strong emissions from other formaldehyde sources. Consequently, ozone-related chemical reactions may not be a major contributor of formaldehyde indoors. Pressed-wood materials bound with urea-formaldehyde resins were likely among the main indoor sources of formaldehyde. Particleboard, medium-density fiberboard (MDF) and hardwood plywood have high formaldehyde emission rates and are widely used in residences for cabinetry, furniture and house construction (Gupta et al. 1982; Kelly et al. 1999; Mølhave et al. 1999).

4.2.2. Influencing Factors

Various factors that affect formaldehyde emissions from new pressed-wood products (PWP) or new house construction materials were evaluated. These factors included ventilation rate (Myers 1984a; Gilbert et al. 2006), indoor temperature (Matthews et al. 1984; Meyer and Hermanns 1985a; Myers 1985), indoor relative humidity (Matthews et al. 1984; Myers 1985), building type (Sexton et al. 1989) and house age (Dingle and Franklin 2002; Stock and Mendez 1985). In general, these factors did not explain much of the variance in C_{in} as shown in Figure 4. The evaluated factors may have had limited influence on mechanisms that control the long-term release of formaldehyde from aging pressed-wood products. When these materials are new, high emissions are due mostly to the evaporation of free formaldehyde in urea-formaldehyde resins as well as the breakdown of easily hydrolyzed chemical bonds. Emissions of free formaldehyde are significantly affected by temperature and relative humidity. As these sources are depleted, formaldehyde is generated from further hydrolysis of the polymeric structure of urea-formaldehyde resins. This mechanism is long-lasting, involves low

emission rates, and is likely to be controlled by diffusion from within the material to its surface (Gammage and Gupta 1984; Nelms et al. 1986).

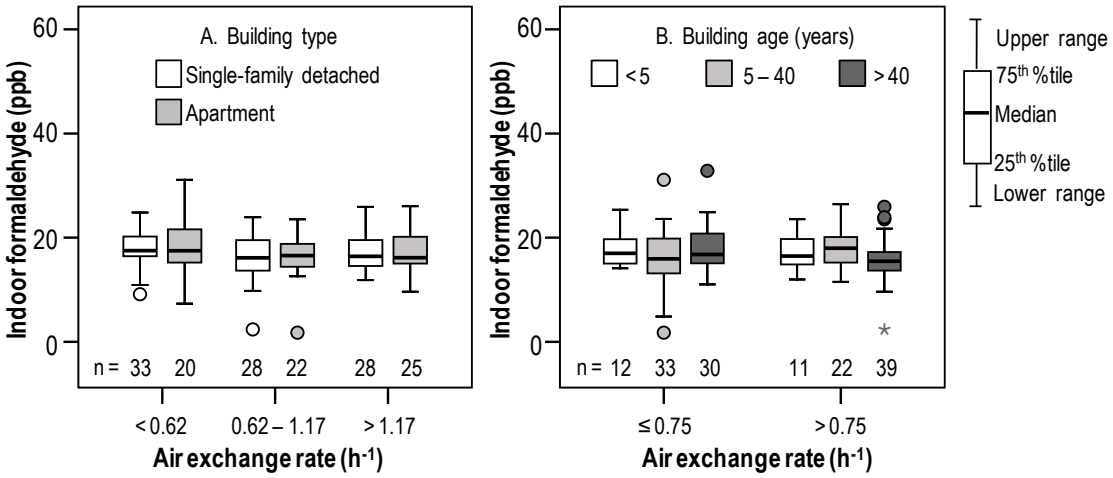

Figure 4. Indoor concentrations of formaldehyde for various air exchange rate categories and building types (*A*) and building ages (*B*).
'o' indicates values between 1.5 and 3 times the interquartile range from the 25th or 75th percentiles.

4.2.3. Mitigation Strategies

The mitigation strategies explored include higher ventilation rates and source control. Previous investigations indicate that increases in AER can lower C_{in}, but these studies involved chamber tests with new pressed-wood materials (Myers 1984a) or tightly-built homes with mean ventilation rates of 0.2 h^{-1} (Gilbert et al. 2006). The present analysis indicated no association between ventilation and C_{in} for collective homes in the RIOPA data base, perhaps because the mitigating effects of AER are influenced by material aging and building tightness as suggested by Meyer and Hermanns (1985b). This lack of association is consistent with the theoretical rate of change of C_{in} with respect to AER (Equation 2), derived by combining a steady-state equation for indoor concentration assuming a well-mixed house and an emission rate equation that is based

on mass transfer away from a source for which an equilibrium concentration can be defined at the source-air interface.

$$\frac{dC_{in}}{d\lambda} = \frac{-k_g L(C_{eq} - C_{out})}{(\lambda + k_g L)^2} \quad (2)$$

Here, L is the loading factor (m^2/m^3) and denotes the ratio of surface area and space volume, λ is the air exchange rate (h^{-1}), k_g is the mass transfer coefficient (m/h), and C_{eq} is the equilibrium concentration at the surface of the emitting material (ppb). Equation 2 shows that C_{in} should decrease with increases in AER (λ), but $|dC_{in}/d\lambda|$ may diminish as materials age and C_{eq} declines, or in homes with high AERs. This is illustrated in Figure 5, where values of 0.5 m/h for k_g and 0.8 m^2/m^3 for L (CARB 2007) were assumed. $|dC_{in}/d\lambda|$ may also decrease at higher ventilation rates because k_g tends to be positively associated with AER.

The rate of change of indoor concentrations with respect to ventilation was also estimated using field measurements for new and existing homes. Data for tightly-built new houses (mean C_{in} = 42 ppb, mean AER = 0.4 h^{-1}, n = 14) were obtained from Sherman and Hodgson (2004) and Hodgson et al. (2000). Measurements from the RIOPA study were used to represent existing residences (mean C_{in} = 17 ppb, mean AER = 1.7 h^{-1}, n = 179). Results from linear regressions indicate that $dC_{in}/d\lambda$ values for tightly-built new homes (-45.7 ppb×h, p = 0.10) were two orders of magnitude greater than those for existing residences (-0.1 ppb×h, p = 0.8). Additionally, C_{in} showed a higher dependence on AER in new homes (R^2 = 0.2) than in older homes (R^2 = 0.0). These results further suggest that the mitigating effects of ventilation decrease with material aging or in houses with high AER.

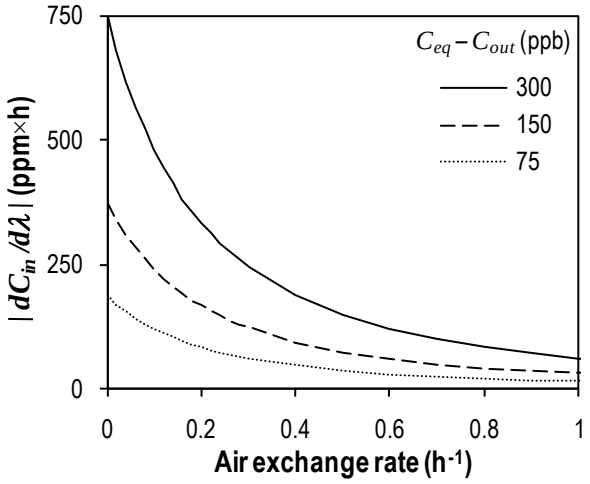

Figure 5. Rate of change of indoor formaldehyde per ventilation rate ($dC_{in}/d\lambda$) as a function of air exchange rate.
Plot is based on Equation 2, k_g = 0.5 m/h, and L = 0.8 m^2/m^3. Abbreviations: C_{eq}, equilibrium concentration; C_{in}, indoor concentration; C_{out}, outdoor concentration; k_g, mass transfer coefficient; L, loading factor; λ, ventilation rate.

Source control strategies to reduce C_{in} include decreasing the amount of pressed-woods materials in homes (Hodgson et al. 2000), utilizing emission barriers on PWPs (Haneto 1986; Hodgson et al. 2002; Kelly et al. 1999), and lowering the formaldehyde content in PWPs (Myers 1984b; CARB 2007). These strategies were concurrently explored in a model that estimated C_{in} using the formaldehyde concentrations from PWPs that will be required in California by 2012 (CARB 2007) based on ASTM Standard Test Method E 1333 (2002). Formaldehyde concentrations were calculated for a 186 m^2 single-family detached home with a ventilation rate of 0.25 h^{-1}, which is the median AER that Offermann et al. (2008) estimated for new California residences. Table B2 describes the parameters that were used in the model; these include emission factors for particleboard, medium-density fiberboard and hardwood plywood; loading factors for various house components (e.g., cabinetry, doors); and emission reduction factors from barriers. Furniture was not included because related loading factors were not available in

the literature; however, furniture can be a very important source given that it is more easily and commonly reintroduced in homes. The estimated indoor formaldehyde concentration was 73 ppb. Although our estimates are high-end values that will diminish with time, it is not evident that C_{in} will meet the REL of 7 ppb (OEHHA 2008), because our calculations did not include furniture made with PWPs and various consumer goods that emit formaldehyde. Nonetheless, we estimated a considerably higher C_{in} of 140 ppb using the 2002 industry average concentrations for PWPs, indicating a dramatic reduction due to regulations in California.

4.3. INDOOR BTEX AND MTBE CONCENTRATIONS FROM PARKED CARS

For this section of the dissertation, the majority of the homes were located in Houston (n = 55), followed by Los Angeles (n = 38) and Elizabeth (n = 21). The residences were either single-family detached (n = 99) or manufactured (n = 15) homes. Indoor and outdoor concentrations for BTEX and MTBE are summarized in Table C1, The percentage of measurements that were above their respective MDLs is also listed in Table C1. Toluene had the lowest percentage of indoor (61%) and outdoor (34%) concentrations greater than the MDL probably because of high toluene background levels in the charcoal pads of Organic Vapor Monitors (Chung et al. 1999). More than 79% of the indoor concentrations and 47% of the outdoor concentrations for the remaining compounds were above their respective MDLs, with the exception of indoor ethylbenzene concentrations in Elizabeth (63%). Median indoor concentrations were typically higher than outdoors; however, statistically significant differences for each compound varied by city probably because of the proximity of sources to the living area and meteorological conditions that affected ventilation-related household activities.

4.3.1. Sources

Various sources of toluene, ethylbenzene and xylenes can be found indoors, such as paints, cleaners, detergents, adhesives, paint thinners and oils/lubricants (DRI 2006; Sack et al. 1992; Nazaroff and Weschler 2004). However, in each of the three cities, indoor MTBE concentrations showed statistically significant positive correlations with C_{in} for BTEX ($0.45 \leq r_s \leq 0.65$). The only exception occurred in Los Angeles, where the association between MTBE and toluene had a *p*-value of 0.07. These correlations indicate that C_{in} for BTEX partly originated from gasoline-related sources, and in particular motor vehicles, because MTBE is a tracer for gasoline. It is very unlikely that MTBE originated from contaminated water because this compound has a high solubility in water and a relatively low Henry's law constant (1.6 mol/(kg×bar) at °C 25 (Sander 2010).

Statistical comparisons between C_{in} and C_{out} were used to examine source location. Table C1 indicates that in the Houston homes C_{in} was statistically higher than C_{out} for all BTEX components. This suggests that sources were within or close to the living area, which was the case for 93% of the households that reported having a parked vehicle nearby during the study. Residences in Elizabeth had indoor and outdoor MTBE concentrations that were not statistically different; only 5% of these homes had cars. However, C_{in} for benzene, toluene and the xylenes were statistically higher than C_{out}, which implies that indoor sources for these VOCs were dominant over ambient mobile sources. In California, C_{in} and C_{out} were statistically similar for all compounds but MTBE ($C_{out} > C_{in}$, $p \leq 0.05$) and toluene ($C_{in} > C_{out}$, $p \leq 0.05$). This indicates that outdoor gasoline-related sources were driving indoor concentrations for every contaminant but toluene, even though 47% of the residences had a vehicle next to the occupied space.

City-specific differences in the percentage of homes that had parked cars during the sampling period may explain why Houston generally had the highest median C_{in} values for all VOCs, whereas Elizabeth usually had the lowest concentrations. The effect of nearby vehicles is reinforced by the fact that car emission standards were the same in Texas and New Jersey during the RIOPA study; both followed U.S. EPA regulations. In contrast, California had stricter emission specifications with regards to non-methane hydrocarbons, which may partly explain the lower indoor BTEX and MTBE concentrations in the Los Angeles homes even though about half of these residences had cars next to the living quarters.

As described in Table C2, variations in ventilation rates also likely contributed to differences in indoor BTEX and MTBE concentrations among cities. Low AERs in Houston (median = 0.48 h^{-1}) limited the dilution of contaminants generated close or within the occupied space with fresh air, whereas high AERs in Los Angeles and Elizabeth (median = 1.1 h^{-1} for both cities) increased the contribution from ambient sources to C_{in}. Discrepancies in AER were likely affected by how households conditioned the indoor space. The mean (median) percent of the sampling period in which households reported to have conditioned the air was 50% (50%) in Houston homes, while much lower values of 4.4% (0%) and 7.6% (0%) were observed in Los Angeles and Elizabeth, respectively. Conversely, the percent of the monitoring time in which windows were reported to have been open was much lower in Houston (mean = 11%, median = 0%) than in Elizabeth (mean = 18%, median = 0%) and Los Angeles (mean = 39%, median = 26%).

The influence of parked cars and ventilation rates on indoor concentrations of BTEX and MTBE were further evaluated. Because of the small sample sizes, data from Los Angeles, Elizabeth and Houston were combined. Residences with vehicles next to

the living area during the sampling period (n = 70) had indoor concentrations that were statistically higher than in homes without such sources (n = 44) for all VOCs but toluene and m&p-xylene. The ratio of median C_{in} values in homes with and without cars ranged from 1.1 (m&p-xylene) to 2.0 (benzene).

The increase in indoor concentrations due to indoor sources was estimated by subtracting C_{out} from C_{in} (ΔC). This increase was statistically higher in homes with cars than in residences without automobiles for all pollutants but MTBE (p = 0.12); variations in the proximity of parked vehicles may have influenced the MTBE results. Median ΔC values ranged from -0.01 $\mu g/m^3$ (MTBE) to 4.7 $\mu g/m^3$ (toluene) when cars were present, and from -0.37 $\mu g/m^3$ (MTBE) to 0.71 $\mu g/m^3$ (toluene) in homes without automobiles. Ventilation rates in homes with vehicles (median = 0.59 h^{-1}) were statistically lower than in residences without cars (median = 1.0 h^{-1}).

4.3.2. Influencing Factors

The effects of source proximity was investigated by examining the six scenarios illustrated in Figure 6: single-family detached (SFD) homes with cars in the attached garage (Scenario 1; n = 14), detached garage (Scenario 2; n = 7), or adjacent carport (Scenario 3; n = 34); manufactured homes with cars in adjacent carports (Scenario 4; n = 15); SFD homes with attached garages but no cars (Scenario 5; n = 8); and SFD homes without both attached garages and cars (Scenario 6; n = 36). Residences in scenarios 3 and 4 were not combined because their indoor concentrations were statistically different. In general, C_{in} for BTEX compounds and MTBE were statistically significantly correlated ($0.34 \leq r_s \leq 0.86$) in all of the studied scenarios, but in cases 4 and 5 where indoor BTEX concentrations appear to have been dominated by non-gasoline-related sources. Single-family detached homes with cars in attached garages were the only case where C_{in} was statistically higher than C_{out} for all pollutants. The SFD homes with cars

in attached garages or carports had the highest median C_{in} for all compounds. In contrast, households without both attached garages and vehicles had the lowest median C_{in} values for all VOCs but toluene. Indoor concentrations in homes with cars in attached garages were likely negatively affected by low house ventilation rates (median = 0.5 h^{-1}) as indicated in Table C2. Residences with vehicles in detached garages were the only case where C_{in} and C_{out} were statistically similar for all contaminants, although the small sample size (n = 7) may have influenced the inability to detect a statistical difference.

Increases in indoor concentrations (relative to outdoors) for all compounds and studied scenarios are shown in Figure 7. Single-family detached homes with vehicles in attached garages had the highest median ΔC for benzene (1.2 $\mu g/m^3$), toluene (6.4 $\mu g/m^3$), m&p-xylene (2.6 $\mu g/m^3$) and MTBE (2.7 $\mu g/m^3$), and relatively large values for ethylbenzene (0.69 $\mu g/m^3$) and o-xylene (0.91 $\mu g/m^3$). Additionally, these homes also had the highest median indoor to outdoor concentration ratios (C_{in}/C_{out}): benzene = 2.0, toluene = 2.7, ethylbenzene = 2.1, m&p-xylene = 2.1, o-xylene = 3.3, MTBE = 1.4. The SFD residences with automobiles in carports tended to have the second highest median ΔCs for all VOCs. For the remaining scenarios, excluding homes with detached garages, median ΔC values were greater than zero for BTEX but not MTBE. Residences with detached garages had median ΔC values that were less than zero ($C_{in} < C_{out}$) for all compounds but toluene. Furthermore, these houses had the lowest median C_{in}/C_{out} ratios for BTEX (0.89 to 1.0), and the same median ratios as homes without cars for MTBE (0.92).

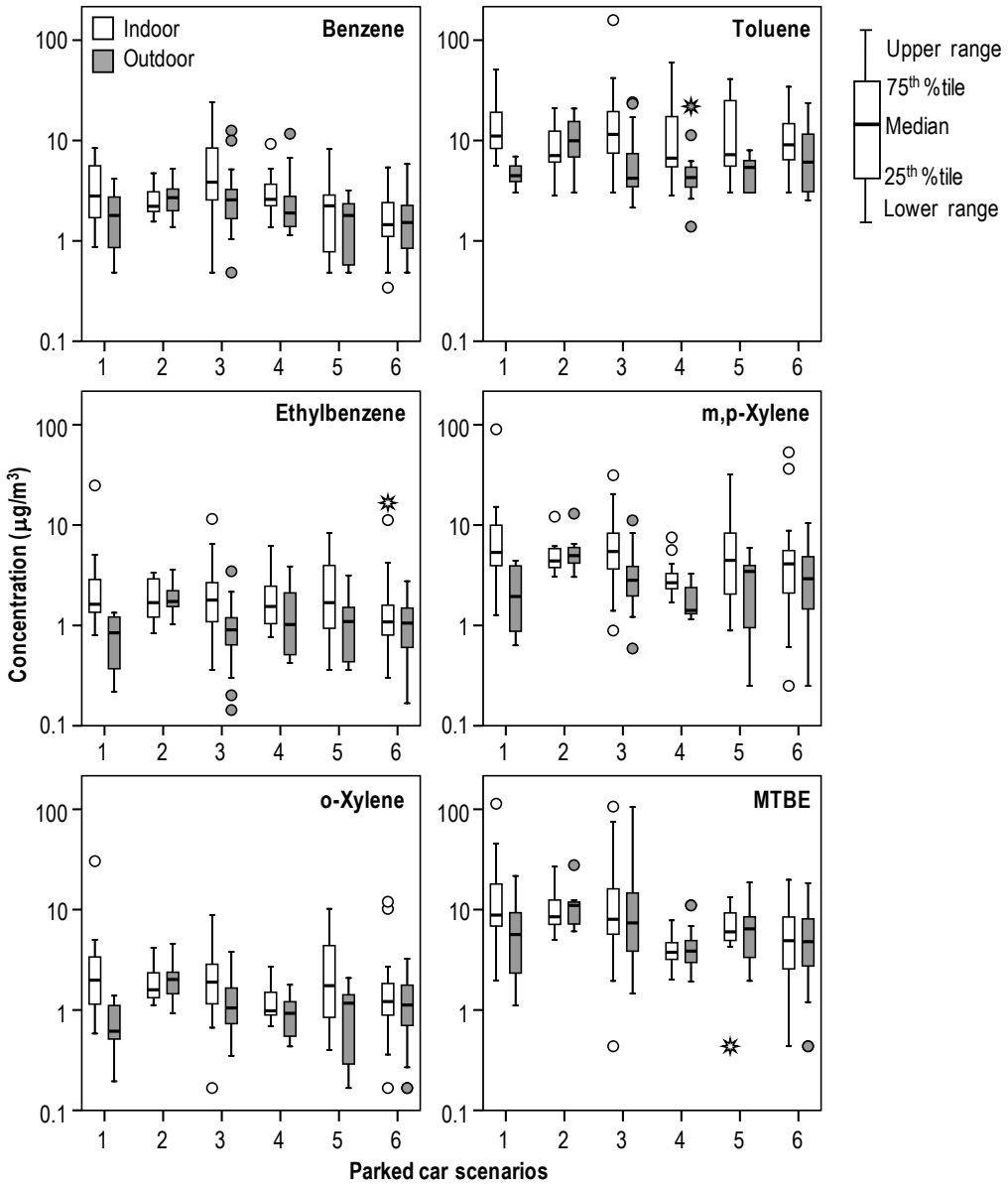

Figure 6. Indoor and outdoor concentrations (µg/m^3) for six scenarios: single-family detached (SFD) homes with cars in the (Scenario 1; *n* =14) attached garage, (Scenario 2; *n* = 7) detached garage, or (Scenario 3; *n* = 34) adjacent carport; (Scenario 4; *n* = 15) manufactured homes with cars in adjacent carports; (Scenario 5; *n* = 8) SFD homes with attached garages but no cars; and (Scenario 6; *n* = 36) SFD homes without both attached garages and cars. 'o' and '*' indicate values between 1.5 and 3, and > 3 box lengths, respectively, from the 25th or 75th percentiles.

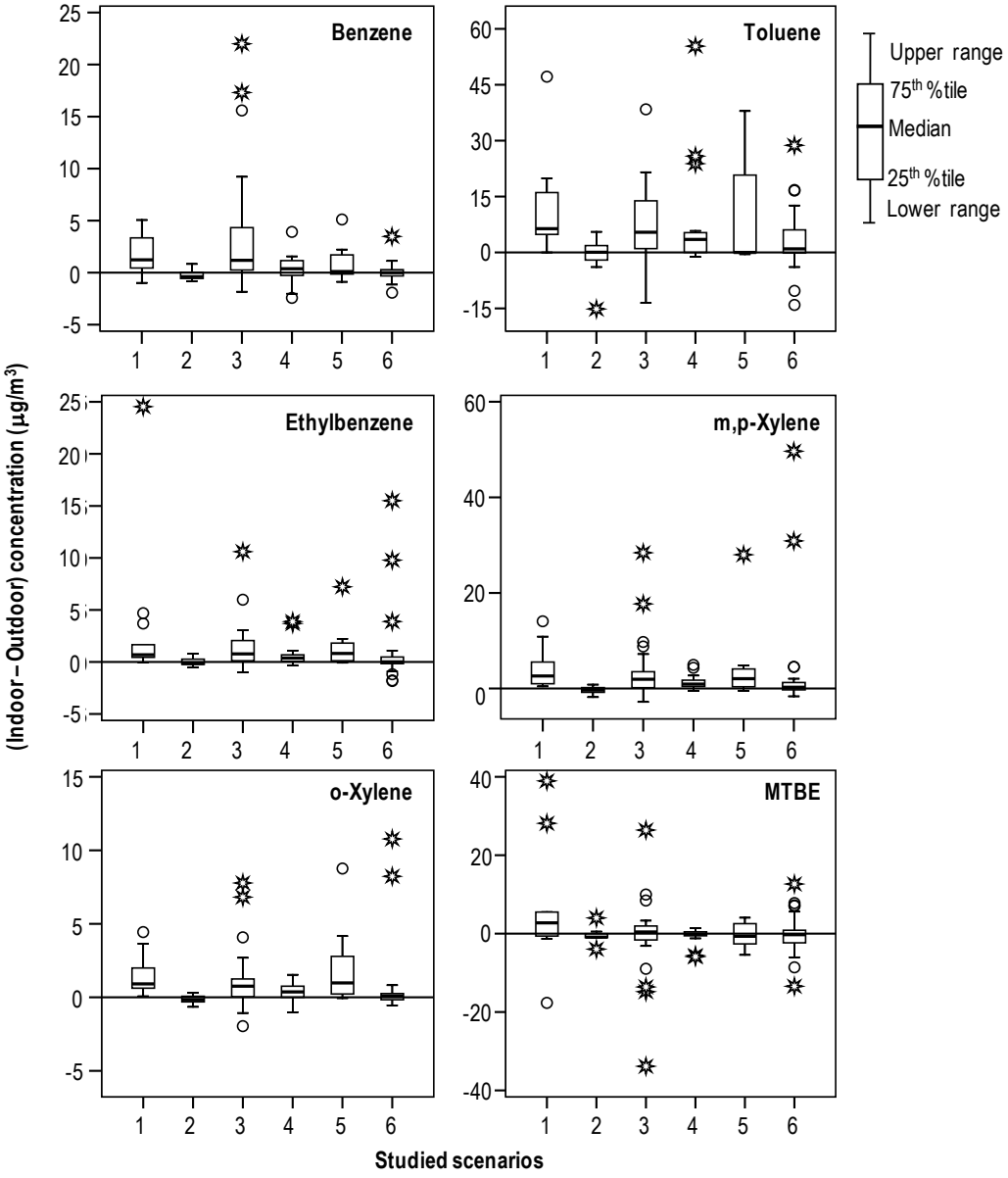

Figure 7. Difference between indoor and outdoor concentrations (µg/m^3) for six scenarios: single-family detached (SFD) homes with cars in the (Scenario 1; *n* =14) attached garage[a], (Scenario 2; *n* = 7) detached garage, or (Scenario 3; *n* = 34) adjacent carport[b]; (Scenario 4; *n* = 15) manufactured homes with cars in adjacent carports; (Scenario 5; *n* = 8) SFD homes with attached garages but no cars; and (Scenario 6; *n* = 36) SFD homes without both attached garages and cars. 'o' and '∗' indicate values between 1.5 and 3, and > 3 times the interquartile range, respectively, from the 25th or 75th percentiles. [a]m&p-xylene = 89 µg/m^3, o-xylene = 30 µg/m^3 and MTBE = 109 µg/m^3 were not included for clarity. [b]Toluene = 155 µg/m^3 and MTBE = 103 µg/m^3 were not included for clarity.

The percent contribution of indoor sources to indoor concentrations (I_{cont}) was calculated by dividing ΔC by C_{in}. This contribution was assumed to be zero in homes where ΔC was negative (i.e., $C_{out} > C_{in}$). The cumulative distribution curve for I_{cont} for four of the six studied scenarios is shown in Figure 8; cases 2 and 5 were omitted because of their small sample size. In general, I_{cont} for most compounds was the highest in residences with automobiles in attached garages; median values ranged from 30% (MTBE) to 52% (ethylbenzene). The SFD homes and manufactured homes with cars in carports had the next highest I_{cont}. Their median values were relatively similar, varying from 5% (MTBE) to 51% (ethylbenzene), and from 0% (MTBE) to 50% (ethylbenzene), respectively. Homes without both attached garages and cars typically had the lowest indoor contributions, median ranged from 0% (MTBE) to 34% (toluene).

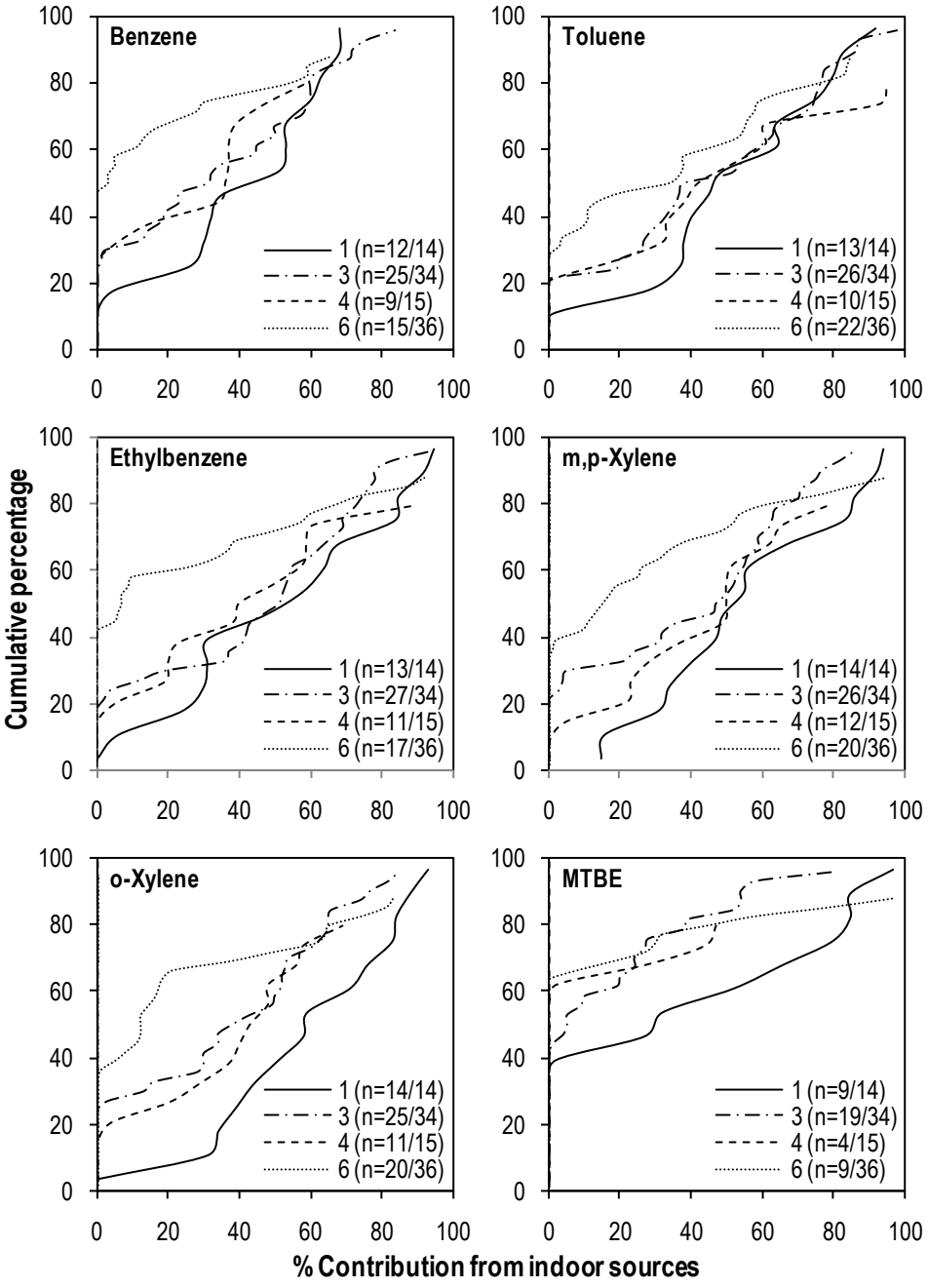

Figure 8. Cumulative distribution functions for the percent contribution from indoor sources to indoor concentrations for four scenarios (n^a): single-family detached (SFD) homes with cars in the (Scenario 1) attached garage, or (Scenario 3) adjacent carport, (Scenario 4) manufactured homes with cars in adjacent carports, and (Scenario 6) SFD homes without both attached garages and cars. Cases 2 and 5 were excluded because of small sample size. [a]Number of homes where indoor concentrations were higher than outdoor concentrations/total number of homes.

The ratio of MTBE to benzene indoor concentrations was used to examine if these compounds originated from gasoline vapors or car exhaust. Low MTBE/benzene ratios indicate that tailpipe emissions are dominant because during combustion the amount of MTBE decreases while benzene is enriched due to toluene and xylene dealkylation (DRI 2006). Alternatively, high MTBE/benzene ratios suggest a significant contribution from evaporative emissions from hot soak, fuel tank "breathing" due to diurnal temperature and barometric changes, and/or fuel system leakage. The Desert Research Institute (DRI 2006) reported ratios for various microenvironments in Houston (freeway = 1.7 – 2.9, in-cabin underground garage = 2.4 – 3.2, in-cabin refueling = 25 – 42, and outdoor refueling = 29 – 56), car exhaust (Houston = 0.44 – 1.4, Los Angeles = 0.43 – 1.1), and liquid gasoline (Houston = 13 – 18, Los Angeles = 12 – 20).

Estimates for MTBE/benzene ratios for the RIOPA homes are shown in Figure 9; here nine houses with indoor benzene measurements that were both below the MDL and less than 1 $\mu g/m^3$ were excluded. Vehicle exhaust appeared to drive C_{in} in about half of the homes given that median ratios for the six studied scenarios ranged from 1.5 to 4.2. For most of the homes with ratios above the median, a mixture of tailpipe and gasoline vapor emissions seemed to have influenced indoor concentrations of gasoline-related VOCs because the six scenarios had 80^{th} percentile ratios that did not exceed 7. Evaporative emissions were substantial in four households where MTBE/benzene values were greater than 11. Two of these were SFD homes with cars in the attached garage. The other two residences did not have cars next to the living area; it can be speculated that these participants likely did not report the presence of gasoline sources indoors given that indoor MTBE concentrations were greater than C_{out} by at least 7.8 $\mu g/m^3$.

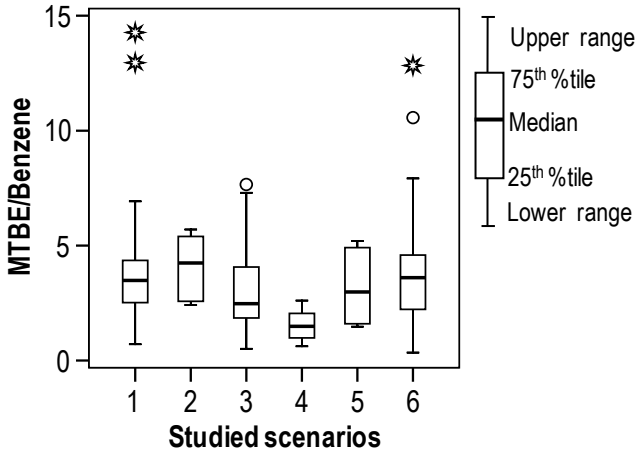

Figure 9. Ratio of MTBE to benzene indoor concentrations for six scenarios: single-family detached (SFD) homes with cars in the (Scenario 1; n =14) attached garage, (Scenario 2; n = 7) detached garage, or (Scenario 3; n = 33) adjacent carport; (Scenario 4; n = 15) manufactured homes with cars in carports; (Scenario 5; n = 6) SFD homes with attached garages but no cars; and (Scenario 6; n = 30) SFD homes without both attahced garages and cars. Twenty homes were excluded where indoor benzene concentrations were both lower than the MDL and less than 1 $\mu g/m^3$. 'o' and '$*$' indicate values between 1.5 and 3, and > 3 times the interquartile range, respectively, from the 25th or 75th percentiles.

To place some of these results into context, the RIOPA data were used to estimate weekly cumulative exposure to benzene in two microenvironments in Houston: homes with vehicles parked in attached garages and cars driven on a freeway with heavy traffic during commute to and from work. Houston was selected because 71% of the RIOPA homes that correspond to the first microenvironment were located in this city. Exposure was calculated by multiplying concentration by the exposure time and dividing the result by the total time spent in the two microenvironments. For homes, the mean ΔC for benzene (2.3 $\mu g/m^3$) was used to better evaluate the effect of parked vehicles and it was assumed that individuals spend 70% of the week in their house (Klepeis 2001). For cars, it was assumed that individuals would be exposed to a mean in-cabin concentration of 6.1 $\mu g/m^3$ (DRI 2006), an average commute time to work of 26 minutes (U.S. Census 2009),

a mean travel time from work equal to the commute time to work, and a five-day work week. Weekly exposure to benzene was 2.2 $\mu g/m^3$ in homes with cars in attached garages, and 0.22 $\mu g/m^3$ in cars during commute to and from work. These results indicate that even though increases in indoor concentrations due to vehicles in attached garages are relatively small, the fact that people spend a large amount of time in their homes can lead to exposures to benzene that are ten times higher than what they may experience in more severe microenvironments that they typically frequent such as heavily congested highways. Additionally, increases in benzene concentration due to vehicles in garages could lead to a mean cancer risk of 17 per million population in Houston, based on an inhalation unit risk factor of 7.8×10^{-6} $m^3/\mu g$ (U.S. EPA 2005). The EPA benchmark for exposure to potential carcinogens is 1 per million.

4.3.3. Mitigation Strategies

ASHRAE Standard 62.2 (2007) describes measures to prevent the migration of pollutants from attached garages into the occupied area in new housing, although these are also applicable to existing residences. These recommendations include (1) sealing vertical and horizontal surfaces shared by these two spaces; (2) avoiding placement of HVAC components in the garage; (3) limiting the total air leakage of HVAC components, especially when located in the garage; and (4) maintaining the living area at a higher pressure than that of the garage. It is not evident that carports are a good alternative to attached garages; SFD homes with cars in carports had relatively high median ΔC values (e.g., benzene = 1.2 $\mu g/m^3$, MTBE = 0.42 $\mu g/m^3$). Conversely, manufactured homes with vehicles in carports had much lower median ΔC values (e.g., benzene = 0.38 $\mu g/m^3$, MTBE = -0.10 $\mu g/m^3$). Various factors could have affected these results such as the location of windows and doors with respect to the carport, the number

of parked cars, and meteorological conditions. Infiltration of pollutants into the living quarters can be limited by tightening the house envelope, specifically close to the carport. In addition to the measures just described, the design of new residences could be improved by incorporating detached garages. Our results indicate that homes with detached garages had minimal increases in indoor concentrations of BTEX and MTBE, which suggests that in addition to cars, non-gasoline-related sources for BTEX may have been stored in the detached garage where they were not as likely to affect the occupied space.

4.4. STUDY LIMITATIONS AND FUTURE RESEARCH

Although the performed cancer risk assessment was a useful tool to identify pollutants of concern and populations at higher risk, the approach has limitations. The calculations underestimate cumulative risk because only 12 gaseous air contaminants were considered due to the few cancer unit risk factors available for the pollutants monitored in RIOPA. Other limitations include uncertainty in the derivation of the cancer potency factors, which involve extrapolations from occupational or animal studies with exposure to high doses. Moreover, cancer risk estimates assume 70-year lifetime exposures, and cumulative effect calculations assume that risks from exposure to various contaminants are additive. Future research should expand the number of contaminants to include others that have been identified as important contributors to cancer risk such as polycyclic organic matter (POM) and 1,3-butadiene (Woodruff et al. 2000). Furthermore, the depth of future work may be increased by including an assessment of possible non-carcinogenic health effects from exposure to lower doses of contaminants (e.g., asthma and irritation of the respiratory system). However, the synergistic impact from various pollutants on non-carcinogenic health effects is not well understood.

This research is based on the analysis of data from the RIOPA study, which is a cross-sectional exposure assessment. A longitudinal evaluation with a representative sample of the population would allow for a more rigorous examination of exposure to HAPs and the various factors that affect these exposures, such as building characteristics and household activity patterns; nonetheless, such an endeavor has limitations. The Center for Disease Control (CDC) has been attempting such an evaluation through the National Health and Nutrition Examination Survey (HNANES). However, the amount of collected information per participant has been constrained given the magnitude of the endeavor.

A number of strategies to reduce exposure are delineated in this dissertation. Some of these were based on prior work, but most of them require further investigation to substantiate their effectiveness. A mitigation mechanism that has not been discussed so far in this dissertation involves intervention strategies, in particular those where investigators from exposure and risk assessments explain to the participants of the study specific findings on their households with regard to pollutant levels, possible health consequences, and ways to reduce exposure. Such an effort could be the most effective way to promote behavioral changes to reduce risk because extensive evidence indicates that exposure to HAPs is highly related to indoor residential contaminants. Furthermore, this intervention strategy could be generalized to launch educational campaigns to reduce risk among disadvantaged populations. Prior work on population subgroups that could be exposed to a greater share of environmental pollution has been mostly based on contaminants emitted by ambient sources. Consequently, educational campaigns are needed to introduce knowledge on indoor air pollution and the fact that personal behavioral practices can have a positive impact.

4.5. LESSONS LEARNED

The lessons learned throughout this research on exposure to HAPs in homes cover the analysis of information from a large database and data collection for an exposure assessment. One of the most important practices in evaluating a large database is to maintain a record of the steps taken during the analysis. For instance, documenting the information stored and examined in data subsets will facilitate future re-examinations on the rationale behind the performed work. This information is very likely to be needed while answering comments from peer reviewers.

As part of the IGERT program on Indoor Environmental Science and Engineering, I completed an internship at the University of Texas Health Sciences Center in Houston. Here, I participated in the Houston Exposure to Air Toxics Study (HEATS), recruiting participants, scheduling visits with participants and collecting data from households. It was apparent from this effort that it is essential for field personnel to be familiar with the cultural practices that prevail in the studied neighborhoods, which will ensure cooperation and participation from individuals. For example, HEATS focused on geographical areas that were highly populated by Hispanics whose first language was Spanish. Being able to communicate with the participants in their native language increased the willingness of individuals to let strangers inside their homes, avoided misunderstandings, and encouraged individuals to allow to be monitored a second time.

5. SUMMARY AND CONCLUSIONS

The main contribution of this dissertation is its in-depth evaluation of data from the RIOPA study, one of the most comprehensive exposure assessments to date. The analyses of data from other assessments have greatly expanded knowledge in the field of exposure; however, sampling techniques have evolved to allow for the collection of more information that can further increase our understanding of exposure to HAPs. Using the wealth of information from the RIOPA study, this dissertation examined air contaminants of concern in residential buildings, the possible sources of these pollutants, and population subgroups with high cancer risks associated with exposure to these contaminants. The effects of building characteristics and household activity patterns on indoor pollution and risk levels were also evaluated to recommend possible strategies to reduce exposure to HAPs. To this end, data from the RIOPA study were utilized to perform a cancer risk assessment using personal concentrations; and to examine associations among (1) personal, indoor, and outdoor concentrations; (2) building characteristics; (3) demographic factors; and (4) household activity patterns.

The following findings emphasize the importance of residential indoor pollution on human exposure to hazardous air pollutants. Results indicate that cumulative cancer risks (*CCR*) from personal concentrations (mean = $1{,}100\times10^{-6}$, median = 480×10^{-6}) are comparable to those estimated in studies that used outdoor concentrations (mean = $1{,}200\times10^{-6}$). The analysis performed with personal concentrations of ten VOCs and two carbonyls that originated from indoor and/or outdoor sources identified formaldehyde, *p*-DCB, acetaldehyde, chloroform and benzene as the main contributors to *CCR*. Indoor sources dominated exposure to the first four compounds, while indoor and/or outdoor sources controlled benzene concentrations. In contrast, risk assessments based on

outdoor concentrations typically identify air contaminants that originate from mobile sources, such as diesel particulate matter, as pollutants of concern. Diesel particulate matter and other carcinogens often measured outdoors were not included in this analysis.

Although both Hispanics and non-Hispanic whites shared the five previously-mentioned compounds as the main contributors to *CCR*, discrepancies in exposure to *p*-DCB, and to a lesser extent to chloroform and benzene, caused statistically significant differences in cumulative risk between these two ethnic groups. *p*-DCB was also responsible for the largest differences in risk within these two groups. This compound appeared to originate from air fresheners in Hispanic homes. Thus, decreasing exposures to *p*-DCB may simply require decreasing the use of these products. These results reinforce the importance of indoor pollution because most studies that have evaluated populations that are at greater risk from air pollution have utilized ambient measurements as surrogates of personal concentrations; these evaluations only provide partial means to reduce exposure and risk levels because their results mostly focus on HAPs from mobile sources.

Formaldehyde may be of more concern for the population at large since this study shows general, similar, chronic exposures to this compound. Indoor exposures to formaldehyde could lead to excess cancers of about 260×10^{-6}, and make this contaminant the highest contributor to *CCR* among individuals with non-extreme risk levels. Pressed-wood materials bound with formaldehyde-urea resins are likely the main indoor source of formaldehyde because they are widespread in homes and these products probably emit this compound throughout their lifetime. Therefore, the removal of pressed-wood materials from residences may be the most effective way to decrease exposures to formaldehyde and cumulative cancer risks for the entire population.

Although BTEX and MTBE are mostly associated with outdoor sources of pollution, vehicles in attached garages can cause indoor concentrations to be statistically higher than ambient concentrations. Indoor concentrations for these VOCs appear to originate from either car exhaust or a combination of evaporative and tailpipe emissions. Individuals could experience weekly cumulative exposures to benzene in homes with vehicles in attached garages that are ten times higher than those experienced while commuting in a car in heavy traffic. Excess cancers and other health risks due to exposure to air contaminants that originate from attached garages may be reduced by parking cars in detached garages, or by following recommendations described in ASHRAE 62.2 (2007) to reduce the infiltration of pollutants from the attached garage to the living area.

Increasing house ventilation rates can decrease exposure to *p*-DCB, acetaldehyde and chloroform. A higher ventilation rate could also lower indoor concentrations of benzene as long as it does not increase the infiltration of pollutants from the attached garage into the occupied space. However, increases in ventilation did not appear to be effective at reducing indoor formaldehyde concentrations of existing homes.

The research findings described in this dissertation underscore that indoor pollution needs to be considered in policies that aim to reduce risk from exposure to hazardous air pollutants for the entire population and population subgroups. In addition to banning sources and reducing emissions from sources, these policies should include educational campaigns because household behavioral patterns can considerably affect exposure to HAPs.

APPENDIX A

Cancer Risk Disparities between Hispanic and Non-Hispanic White Populations: The Role of Exposure to Indoor Air Pollution

(Published in *Environmental Health Perspectives*. 2009. 117(12):1925-1931)

ABSTRACT

Background

Hispanics are the fastest growing minority group in the U.S.; however, there is minimal information on their cancer risks from exposures to hazardous air pollutants (HAPs) and how these compare to those from non-Hispanic Whites.

Methods

We assess the personal exposure and cancer risk of Hispanic and White adults who participated in the Relationships of Indoor, Outdoor, and Personal Air (RIOPA) study. We evaluate twelve of the sampled volatile organic compounds and carbonyls, and identify HAPs of most concern and their possible sources. Furthermore, we examine socio-demographic factors and building characteristics.

Results

Cumulative cancer risks (*CCR*s) for Hispanics (median = 519×10^{-6}, 90^{th} percentile = $3{,}968\times10^{-6}$) and Whites (median = 443×10^{-6}, 90^{th} percentile = 751×10^{-6}) were much greater than the U.S. Environmental Protection Agency (EPA) benchmark of 10^{-6}. Cumulative risks were dominated by formaldehyde and *p*-dichlorobenzene, and to a lesser extent by acetaldehyde, chloroform and benzene. Exposure to all of these compounds but benzene was primarily due to indoor residential sources. Hispanics had statistically higher *CCR*s than Whites ($p \leq 0.05$) because of differences in exposure to *p*-dichlorobenzene, chloroform and benzene. Formaldehyde was the largest contributor to

CCR for 69% of Hispanics and 88% of Whites. Cancer risks for pollutants emitted indoors increased at lower house ventilation rates.

Conclusions

Hispanics appear to be disproportionately affected by certain HAPs from indoor and outdoor sources. Policies that aim to reduce risk from exposure to HAPs for the entire population and population subgroups should consider indoor air pollution.

INTRODUCTION

Evidence suggests that disparities in environmental exposures may disproportionately affect the health of ethnic minorities. Census tracts with higher proportions of Hispanics or African-Americans appear to have higher outdoor levels of hazardous air pollutants (HAPs) than tracts with higher proportions of non-Hispanic Whites (Apelberg et al. 2005; Linder et al. 2008; Morello-Frosch and Jesdale 2006). However, this evidence is mostly based on outdoor measurements and much less is known about exposure to indoor air pollution. Therefore, inhalation exposure assessments are needed to improve knowledge of environmental risk, given that these evaluations involve monitoring personal concentrations in the breathing zone of individuals throughout their daily activities. Such monitoring incorporates the penetration of outdoor pollutants into buildings, as well as important contributions from indoor sources of HAPs and the large fraction of time people spend indoors. The importance of indoor air to overall inhalation exposure is supported by results from various studies, most notably the Total Exposure Assessment Methodology (TEAM; Wallace 1991) and the Relationships of Indoor, Outdoor, and Personal Air (RIOPA; Weisel et al. 2005) studies. These investigations demonstrate that some indoor sources can have greater effects on personal exposure to HAPs than those of outdoor origin.

Results from exposure assessments suggest that minority groups may have high exposures to specific HAPs that could cause significant disparities between these groups and the majority population. Pellizzari et al. (1999) used air pollutant data from the National Human Exposure Assessment Survey (NHEXAS) to determine that minorities had higher personal measurements for lead and benzene than non-minorities, but the authors cautioned that their sample size for minorities was small. Churchill et al. (2001) analyzed exposure to VOCs through blood samples collected in the Third National Health and Nutrition Examination Survey (NHANES III) and indicated that African-Americans and Mexican-Americans were more likely to have elevated levels of *p*-dichlorobenzene (*p*-DCB) than Whites. African-Americans also had higher blood levels of chloroform and tetrachloroethene than Whites. More recently, D'Souza et al. (2009) evaluated HAP data from the 1999-2000 NHANES and concluded that Hispanics and African-Americans had much higher personal concentrations for BTEX (benzene, toluene, ethylbenzene, xylenes), methyl *tert*-butyl ether (MTBE) and *p*-DCB than Whites. However, NHANES did not evaluate exposure to carbonyls, building characteristics such as home ventilation rates, and cancer risks. The remaining investigations in the literature mostly provide insight on *p*-DCB and chloroform as possible pollutants of concern among minorities (Adgate et al. 2004; Sax et al. 2006).

In this paper we evaluate the cancer risks of Hispanics and non-Hispanic Whites due to exposure to HAPs using data from the RIOPA study. In RIOPA, non-smoking residences in Los Angeles County, California (n = 105), Elizabeth, New Jersey (n = 100), and Houston, Texas (n = 106), were monitored. Approximately 48% of adult participants described themselves as Hispanic and 38% as White. We focus on 12 of the sampled airborne volatile organic compounds (VOCs) and carbonyls for which cancer unit risk factors are available, and use personal concentrations to estimate contaminant-specific

cancer risks and cumulative cancer risks. We identify pollutants of most concern and explore their possible origins. We also investigate factors that could contribute to risk disparities by examining associations with demographic and building characteristics since previous investigations reported that these could affect exposure to HAPs (Apelberg et al. 2005; D'Souza et al. 2009; Johnson et al. 2004; Linder et al. 2008).

MATERIALS AND METHODS

Data from the RIOPA study were made available by the Health Effects Institute (HEI 2008). The RIOPA study involved the recruitment of non-smoking adults who resided in Los Angeles County, California, Elizabeth, New Jersey, and Houston, Texas. Participants in Houston and Elizabeth constitute a convenience sample, while the Los Angeles participants were a subset from a randomly selected sample of individuals from another study. Approximately 100 adults volunteered in each city, most of whom worked at home or their workplace was in the same neighborhood as their residences. About 65% of the homes were located in close proximity to major outdoor sources of pollution such as highways in Los Angeles, petrochemical facilities in Houston, and small sources (e.g., dry cleaners) in Elizabeth.

Weisel et al. (2005) provide a detailed description of the RIOPA field and measurement protocols. Briefly, from 1999 to 2001, participants and their homes were monitored during two 48-hour periods that were approximately three months apart. Air contaminants were selected to include HAPs that are categorized by the U.S. Environmental Protection Agency (EPA) as urban air toxics or mobile-source pollutants, as well as compounds from primarily indoor origin. Air samples were collected concurrently in the personal or breathing zone, and inside and outside the house. Sixteen VOCs were monitored using Organic Vapor Monitors (OVM 3500, 3M Company, St. Paul, MN, USA), and ten carbonyls were collected using Passive Aldehyde and Ketones

Samplers (PAKS; Zhang et al. 2001). Concentrations at or below the respective method detection limit (MDL) were censored by replacement with ½ the MDL concentrations. The effects of censoring on the cancer risk assessment were small because at least 50% of the concentrations that contributed most significantly to risk were well above the MDL. Demographic and building characteristics, as well as daily indoor/outdoor activity patterns were collected during each of the sampling sessions with questionnaires and walkthrough surveys. Residential air exchange rates (AERs) were determined using tracer gas decay.

Cancer risks were used to evaluate the relative importance of sampled pollutants. Therefore, this investigation focused on 12 of the sampled HAPs for which estimates of cancer unit risk factors are available as shown in Table 1. Risk factors were primarily obtained from the U.S. EPA (2005); however, estimates from CalEPA (2002, 2005) and Caldwell et al. (1998) were used when not available from the U.S. EPA. Only houses with personal concentrations for all of these 12 compounds in either of the monitoring sessions were considered, reducing the overall sample size from 311 to 246. Estimates of cancer risks for each HAP were derived as:

$$CR = P \times UR \qquad [1]$$

where *CR* is the cancer risk, *P* is the measured personal concentration (μg/m^3), and *UR* is the inhalation cancer unit risk factor and represents the probability of cancer for a 70-year exposure to 1 μg/m^3. The cumulative cancer risk (*CCR*) was calculated by summing the *CR*s from all 12 HAPs (Caldwell et al. 1998).

Table 1. Measured hazardous air pollutants in RIOPA with available cancer unit risk factors.

Compound	WOE		Unit risk (per µg/m^3)	Source[c]
	IRIS[a]	IARC[b]		
Acetaldehyde	B2	2B	2.2×10^{-6}	1
Benzene	A	1	7.8×10^{-6}	1
Carbon tetrachloride	B2	2B	1.5×10^{-5}	1
Chloroform	B2	2B	2.3×10^{-5}	1
Ethylbenzene	NC	2B	2.5×10^{-6}	2
Formaldehyde	B1	1	1.3×10^{-5}	1
Methylene chloride	B2	2B	4.7×10^{-7}	1
MTBE	NC	3	2.6×10^{-7}	3
p-Dichlorobenzene	NC	2B	1.1×10^{-5}	3
Styrene	NC	2B	5.0×10^{-7}	4
Trichloroethylene	NC	2A	2.0×10^{-6}	3
Tetrachloroethylene	NC	2A	5.9×10^{-6}	3

Abbreviations: IARC, International Agency for Research on Cancer; IRIS, Integrated Risk Information System; MTBE, methyl *tert*-butyl ether; WOE, weight of evidence.
[a]IRIS classification: A, known carcinogen; B1, probable carcinogen; B2, probable carcinogen; NC, not classified.
[b]IARC classification: 1, carcinogenic; 2A, probably carcinogenic; 2B, possibly carcinogenic; 3, not classifiable as to carcinogenicity to humans.
[c]Sources: 1, IRIS (U.S. EPA, 2005); 2, CalEPA (2002); 3, CalEPA (2005); 4, Caldwell et al. (1998).

Several conventions were followed throughout this research. Measurements from a household where someone smoked during a sampling period were excluded (n = 8). Information from the two sampling sessions was consolidated into a single dataset. Air concentrations for each pollutant and air exchange rates were averaged when the household was monitored twice. In most instances, demographic data from the first visit were selected when information from the first and second sessions were not in agreement. In the case of income, the midpoints of disparate income ranges were averaged.

Nonparametric statistical analyses were utilized because pollutant concentrations typically had positively skewed distributions. The Wilcoxon rank-sum test was used to evaluate differences between two independent samples, such as personal concentrations

from Hispanics and Whites. Similarly, the Kruskal-Wallis test was employed with three independent variables. The Wilcoxon sign-rank test was used to assess differences between paired samples, such as concurrent personal and indoor concentrations. Results were considered statistically significant at $p \leq 0.05$. SPSS (version 15.0, SPSS Inc.) was employed for these analyses.

RESULTS

Cumulative cancer risks (*CCR*s) for all participants in the RIOPA study ($n = 238$) were greater than 10^{-4}. Mean, median and 90th percentile *CCR*s were $1{,}126\times10^{-6}$, 485×10^{-6} and $1{,}675\times10^{-6}$, respectively, after excluding two unusually high measurements for chloroform (1,224 μg/m^3) and tetrachloroethylene (1,340 μg/m^3). The principal contributors to the mean *CCR* were *p*-DCB (60%) and formaldehyde (26%). For individuals with the highest risks (i.e., top 10th percentile), *p*-DCB accounted for 91% of the mean *CCR*.

Differences between Hispanics and Non-Hispanic Whites

The percentage of Hispanic participants was the largest in Elizabeth (EL; 78%), followed by Houston (HO; 55%) and Los Angeles (LA; 35%) as indicated in Table 2. Figure 1 shows that Hispanics and Whites had cancer risks for nine of the twelve studied pollutants that were higher than the U.S. Environmental Protection Agency (EPA) benchmark of 10^{-6}, but there were differences in risk between the two ethnic groups that varied by city. *CCR* was higher among Hispanics than Whites in EL ($p \leq 0.05$) and HO ($p \leq 0.01$). The median *CCR* of Hispanics in EL, 506×10^{-6}, was 1.2 times higher than that for Whites. This ratio increased to 1.6 in HO, where Hispanics had a median cumulative risk of 723×10^{-6}. For LA, the *CCR* was about 438×10^{-6} for both ethnic groups and similar to that of Whites in EL and HO. The main contributors to *CCR* were

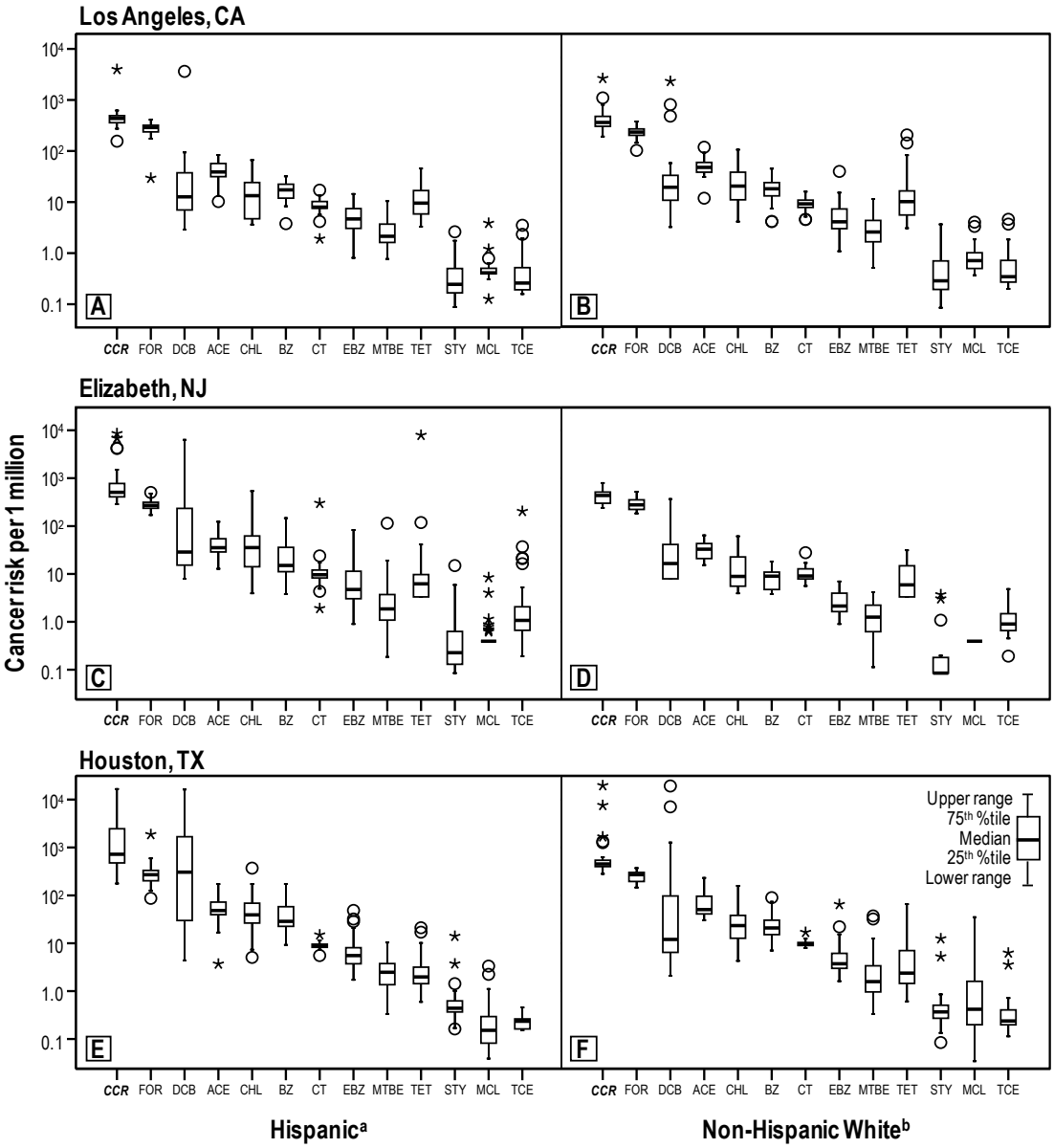

Figure 1. Distributions of cancer risks based of Hispanic and Whites in Los Angeles (*A* and *B*), Elizabeth (*C* and *D*), and Houston (*E* and *F*). 'o' and '*' indicate values between 1.5 and 3, and > 3 times the interquartile range, respectively. Hispanic: Los Angeles, $n = 23$; Elizabeth, $n = 54$; Houston, $n = 44$. Non-Hispanic White: Los Angeles, $n = 43$; Elizabeth, $n = 15$; Houston, $n = 36$. Abbreviations: ACE, acetaldehyde; BZ, benzene; CCR, cumulative cancer risk; CHL, chloroform; CT, carbon tetrachloride; DCB, *p*-dichlorobenzene; EBZ, ethylbenzene; FOR, formaldehyde; MCL, methylene chloride; MTBE, methyl tert-butyl ether; STY, styrene; TCE, trichloroethylene; TET, tetrachloroethylene.

Table 2. Descriptive summary of personal concentration (μg/m^3), by city and ethnic group.

Compound	Hispanic							Non-Hispanic White							
	n (%)[a]	Mean	SD	Med	%>MDL	P&I[b]	P&O[b]	*n* (%)[a]	Mean	SD	Med	%>MDL	P&I[b]	P&O[b]	H&W[c]
Los Angeles															
Acetaldehyde	23 (61)	19.8	8.58	17.7	100	I*	P**	43 (63)	24.9	10.1	23.1	100		P**	W*
Benzene	23 (61)	2.31	0.91	2.23	89			43 (77)	2.56	1.32	2.33	87			
Carbon tetrachloride	23 (61)	0.59	0.21	0.53	92		O*	43 (77)	0.61	0.18	0.59	93		O**	
Chloroform	23 (61)	0.83	0.76	0.58	76		P*	43 (77)	1.38	1.29	0.90	87	I*	P**	W*
Ethylbenzene	23 (61)	2.29	1.46	1.87	89		P**	43 (77)	2.30	2.62	1.49	88		P**	
Formaldehyde	23 (61)	21.4	6.19	22.2	100		P**	43 (63)	21.7	5.44	21.3	100	P*	P**	
Methylene chloride	23 (61)	1.33	1.56	0.87	51		P**	43 (77)	1.63	1.38	1.22	61		P**	W*
MTBE	23 (61)	12.7	10.2	8.21	95			43 (77)	11.1	7.84	8.75	99	P**		
p-Dichlorobenzene	23 (61)	16.5	68.2	1.15	46		P*	43 (77)	12.1	46.2	1.78	54		P**	
Styrene	23 (61)	1.04	1.29	0.49	65		P*	43 (77)	1.04	1.40	0.44	58		P**	
Trichloroethylene	23 (61)	2.30	1.99	1.61	86		P*	43 (77)	3.81	7.47	1.66	86		P**	
Tetrachloroethylene	23 (61)	0.31	0.42	0.13	43			43 (77)	0.28	0.39	0.13	45			
Elizabeth															
Acetaldehyde	54 (69)	18.6	8.2	16.1	100	P**	P**	15 (67)	15.6	6.71	14.9	100		P**	
Benzene	54 (81)	3.64	4.21	1.93	78		P**	15 (87)	1.16	0.59	1.15	57		P**	H**
Carbon tetrachloride	54 (81)	1.03	2.64	0.64	94			15 (87)	0.74	0.39	0.60	82			
Chloroform	54 (81)	3.18	4.80	1.55	78	P**	P**	15 (87)	0.79	0.82	0.39	54		P**	H**
Ethylbenzene	54 (81)	3.83	6.20	1.89	81		P**	15 (87)	1.12	0.72	0.85	64		P**	H**
Formaldehyde	54 (69)	21.9	5.82	20.8	100		P**	15 (67)	23.0	7.54	21.4	100	P*	P**	
Methylene chloride	54 (81)	1.41	2.53	0.84	9			15 (87)	0.84	0.00	0.84	0			H*
MTBE	54 (81)	20.2	60.0	7.16	93	P**	P**	15 (87)	5.94	4.57	4.82	82			H*
p-Dichlorobenzene	54 (81)	44.1	123	2.61	63		P**	15 (87)	4.54	8.45	1.51	36		P**	H*
Styrene	54 (81)	1.89	4.61	0.46	54	P*	P**	15 (87)	1.21	2.32	0.17	29		P*	H*
Trichloroethylene	54 (81)	26.4	182	1.06	44		P**	15 (87)	1.80	1.55	1.01	57	P**	P**	
Tetrachloroethylene	54 (81)	3.35	14.0	0.54	74		P**	15 (87)	0.73	0.73	0.45	71		P*	

Table 2. Continuation

Compound	Hispanic							Non-Hispanic White							
	n (%)[a]	Mean	SD	Med	%>MDL	P&I[b]	P&O[b]	*n* (%)[a]	Mean	SD	Med	%>MDL	P&I[b]	P&O[b]	H&W[c]
Houston															
Acetaldehyde	44 (64)	25.9	14.5	21.9	97		P**	36 (39)	35.6	24.8	23.0	100	I**	P**	
Benzene	44 (93)	5.77	4.55	3.68	100		P**	36 (94)	3.46	2.29	2.68	100	P*	P**	H**
Carbon tetrachloride	44 (93)	0.60	0.10	0.58	99			36 (94)	0.66	0.11	0.64	100		P**	W**
Chloroform	44 (93)	2.67	2.81	1.70	96		P**	36 (94)	1.47	1.38	1.02	89		P**	H**
Ethylbenzene	44 (93)	3.35	3.54	2.21	100	P**	P**	36 (94)	2.79	4.29	1.49	100	P**	P**	H*
Formaldehyde	44 (64)	23.8	19.9	20.7	97		P**	36 (39)	19.9	4.75	20.8	100		P**	
Methylene chloride	44 (93)	0.77	1.29	0.32	74	P*	P**	36 (94)	4.93	12.9	0.89	87		P**	W**
MTBE	44 (93)	11.5	8.78	9.54	98	P**	P**	36 (94)	16.9	30.7	6.09	99		P**	
p-Dichlorobenzene	44 (93)	162	312	27.7	84	I*	P**	36 (94)	75.5	306	1.09	66	P*	P**	H**
Styrene	44 (93)	1.76	4.20	0.88	92		P**	36 (94)	1.68	4.32	0.74	87	P**	P**	H**
Trichloroethylene	44 (93)	0.57	0.69	0.34	79	P**	P**	36 (94)	1.72	2.87	0.40	80	P*	P**	
Tetrachloroethylene	44 (93)	0.12	0.04	0.12	25		P*	36 (94)	0.27	0.57	0.12	37	P*		

Abbreviations: MDL, method detection limit; Med, median; MTBE, methyl *tert*-butyl ether.

[a]Number of participants (percentage of participants that were sampled twice).

[b]P: personal concentrations were statistically higher than indoor or outdoor concentrations. I: indoor concentrations were statistically higher than personal concentrations. O: outdoor concentrations were statistically higher than personal concentrations.

[c]H: measurements were statistically higher among Hispanics than Whites. W: measurements were statistically higher among Whites than Hispanics.

*$0.01 < p \leq 0.05$.

**$p \leq 0.01$.

formaldehyde, *p*-DCB, acetaldehyde, chloroform and benzene, accounting for at least 83% of the cumulative risk among Hispanics and 92% among Whites in each of the cities. The contribution among Hispanics increased to 95% after an unusually large tetrachloroethylene concentration in EL (1,340 $\mu g/m^3$) was excluded.

Given the skewed distribution of the *CCR*s in Figure 1, we analyzed the cumulative risk tertiles. Figure 2 shows the average contribution of each HAP to the mean of *CCR* tertiles. Most of the 1st and 2nd tertiles for all of the studied scenarios were similar with mean cumulative values ranging from 281×10^{-6} to 524×10^{-6}. Formaldehyde contributed 55 to 77% of the mean *CCR*, while acetaldehyde, benzene, chloroform and *p*-DCB accounted for 18 to 36%. The 2nd tertile for Hispanics in HO differed by having a much higher mean value of 771×10^{-6}, where *p*-DCB was the main contributor (39%) over formaldehyde (35%). Mean cumulative risks increased from the 2nd to 3rd tertiles more for Hispanics (factor of 2.3 to 7.2 across all cities) than for Whites (factor of 1.3 to 6.4 across all cities). Increases in risk in LA and EL were primarily due to *p*-DCB, which accounted for approximately 53% of the mean *CCR* among Hispanics ($CCR_{LA} = 969\times10^{-6}$, $CCR_{EL} = 2{,}437\times10^{-6}$) and 28% among Whites ($CCR_{LA} = 889\times10^{-6}$, $CCR_{EL} = 604\times10^{-6}$). In Houston, *p*-DCB was responsible for 88% of the mean *CCR* for Hispanics ($CCR = 5{,}537\times10^{-6}$) and 64% for Whites ($CCR = 2{,}964\times10^{-6}$). Formaldehyde was the second most important pollutant for all of the 3rd tertiles, contributing 7 to 34% of the mean *CCR* among Hispanics and 10 to 60% among Whites. *CCR*s for Hispanics and Whites in the top two tertiles remained statistically different (i.e., Hispanics > Whites) after *p*-DCB was excluded from the cumulative risk calculations in EL and HO.

Personal concentrations for the two ethnic groups are summarized in Table 2. Among the main contributors to *CCR*, Hispanics in EL and HO had personal exposures that were statistically higher than those for Whites for benzene, chloroform and *p*-DCB.

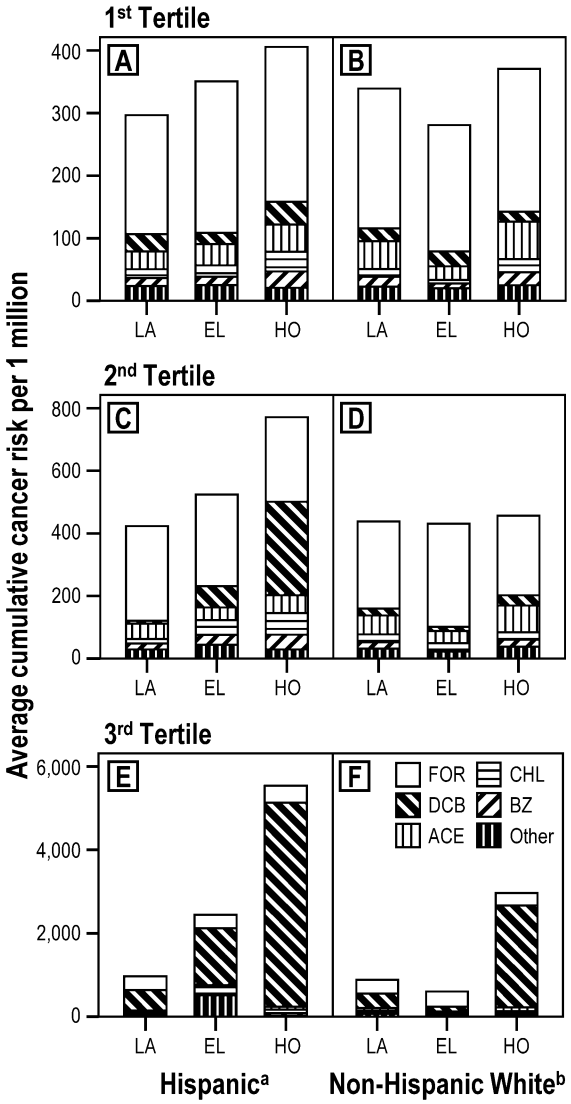

Figure 2. Average of 1st (*A* and *B*), 2nd (*C* and *D*), and 3rd (*E* and *F*) *CCR* tertiles for Hispanics (*A*, *C* and *E*) and non-Hispanic Whites (*B*, *D* and *F*). Every tertile shows the average contribution of each HAP. Hispanic: LA: $n = 23$, EL: $n = 54$, HO: $n = 44$. Non-Hispanic White: LA: $n = 43$, EL: $n = 15$, HO: $n = 36$. Abbreviations: ACE, acetaldehyde; BZ, benzene; CHL, chloroform; DCB, *p*-dichlorobenzene; EL, Elizabeth; FOR, formaldehyde; HO, Houston; LA, Los Angeles; Other, carbon tetrachloride, ethylbenzene, methylene chloride, methyl *tert*-butyl ether, styrene, tetrachloroethylene, trichloroethylene.

Whites in LA had statistically higher exposures than Hispanics for acetaldehyde and chloroform, but these discrepancies were not large enough to cause statistical differences

in *CCR*. Although formaldehyde did not contribute to risk disparities, it is worth noting that exposures were similar for all groups in all cities, suggesting chronic effects throughout the entire population. Formaldehyde had a mean and median personal concentration of 21 $\mu g/m^3$ (cancer risk = 276×10^{-6}), and the lowest coefficient of variance (28%) for all the evaluated HAPs after the exclusion of a relatively high personal measurement (144 $\mu g/m^3$) that was inconsistent with the corresponding indoor concentration (26 $\mu g/m^3$).

Differences within Hispanics and Non-Hispanic Whites

The demographic factors described in Table 3 were analyzed to identify subgroups that may be at greater risk. Tests of statistical differences of cancer risks (*CR*) were performed within ethnic groups. Because of the small sample sizes, data from LA, EL and HO were combined. The following results focus on the five HAPs that were the main contributors to *CCR*. The analysis indicates that Hispanic women had greater risk from exposure to chloroform (median = 36×10^{-6}) than did men ($p \leq 0.01$), and their median values differed by a factor of 2.7. Hispanics who earned less than $25,000 had median risks for *p*-DCB (95×10^{-6}) that were 4.1 times higher than those with greater incomes ($p \leq 0.01$). Furthermore, Hispanics whose homes were less than 1 km from major outdoor sources of HAPs had median *CR*s for benzene (25×10^{-6}) and *p*-DCB (110×10^{-6}), as well as *CCR* (642×10^{-6}) that were statistically higher than those without these outdoor sources nearby. The apparent relationship between risk from *p*-DCB and proximity to outdoor sources is probably due to the prevalence of indoor sources of this pollutant in lower income homes. Fifty-eight percent of Hispanics who lived close to ambient sources had annual family earnings that were less than $25,000. Moreover, these individuals tended to have high personal concentrations of *p*-DCB, while major outdoors

sources of this contaminant were not present. Significant differences in cancer risk from demographic factors were not observed among Whites.

Some of the building characteristics listed on Table 3 also influenced cancer risk. Just as with demographics, the data from the three cities were pooled for the two ethnic groups. Home age was evaluated because it could be an indicator of the emission strength for certain HAPs from new building materials, as well as greater ventilation rates for older buildings. Hispanics whose houses were less than 15 years old had statistically higher median risks for acetaldehyde (48×10^{-6}) and chloroform (51×10^{-6}) than those who lived in older homes. Similar trends were observed with Whites in newer homes for chloroform (40×10^{-6}), benzene (21×10^{-6}) and *CCR* (534×10^{-6}). In general, there was a negative association between risk and the three air exchange rate (AER) ranges that were assessed (< 0.5, 0.5 – 1.0, > 1.0 hr^{-1}). Hispanics in homes with low ventilation rates (< 0.5 hr^{-1}) had statistically higher median risks for acetaldehyde (54×10^{-6}), chloroform (61×10^{-6}) and *p*-DCB (141×10^{-6}), as well as *CCR*s (725×10^{-6}) than those in houses with high AERs (> 1.0 hr^{-1}). Air exchange rates were most influential on exposure to *p*-DCB, with a median risk ratio of 6.7 between homes with low and high AERs. Ventilation rates affected Whites in a similar manner, with subjects in tighter houses (< 0.5 hr^{-1}) having median *CR* values for chloroform (24×10^{-6}) and *CCR* (505×10^{-6}) that were statistically higher than those in homes with AERs greater than 1 hr^{-1}. There may have been some overlap between the positive associations of house age and ventilation rate and the risks from exposure to certain compounds generated indoors such as chloroform. This is consistent with the observed small increases in median AERs with building age. Nevertheless, house age may not be a good indicator of ventilation rate given that each of the studied age categories had comparable 5^{th} and 95^{th} percentile AER values.

Table 3. Selected household characteristics by city and ethnic group (n(%))[a].

Description	Los Angeles		Elizabeth		Houston	
	Hispanic	White	Hispanic	White	Hispanic	White
Gender						
Male	8 (63)	14 (71)	9 (78)	4 (75)	4 (75)	9 (33)
Female	15 (60)	29 (59)	45 (67)	11 (64)	40 (62)	27 (41)
Income						
< $25,000	7 (29)	8 (75)	23 (70)	4 (50)	30 (53)	5 (40)
$25,000 – 49,999	4 (75)	17 (53)	6 (100)	3 (67)	10 (90)	14 (50)
$50,000 – 74,999	10 (70)	7 (86)	5 (80)	4 (100)	3 (100)	10 (30)
> $75,000	2 (100)	11 (54)	3 (100)	3 (67)	0	6 (33)
Don't know	0	0	13 (46)	1 (0)	1 (0)	1 (0)
Refused to answer	0	0	3 (67)	0	0	0
Building Type						
Mobile/Trailer	1 (100)	2 (50)	0	0	22 (50)	2 (0)
Single-family detached	15 (60)	22 (59)	9 (78)	7 (57)	20 (16)	34 (41)
Single-family attached	0	3 (0)	4 (50)	1 (100)	0	0
Apartment	7 (71)	16 (81)	38 (71)	7 (71)	2 (50)	0
Building Age (years)						
< 5	4 (100)	12 (83)	2 (100)	0	4 (50)	0
5 – 15	0	2 (50)	4 (75)	0	7 (29)	7 (29)
> 15	17 (47)	29 (55)	24 (67)	14 (71)	24 (79)	27 (44)
Don't know	2 (100)	0	24 (67)	1 (0)	9 (56)	2 (0)
Building AER (hr^{-1})						
< 0.5	1 (0)	9 (100)	5 (80)	2 (100)	17 (53)	22 (73)
0.5 – 1.0	9 (56)	15 (80)	21 (90)	4 (50)	14 (71)	12 (83)
> 1.0	13 (62)	19 (95)	26 (81)	8 (62)	8 (62)	2 (50)
< 1 km from industry	3(67)	6 (67)	20 (65)	9 (78)	42 (64)	32 (41)
Total households	23	43	54	15	44	36

[a]Data are presented as number of participants (percentage of participants sampled twice).

Sources of HAPs

The possible origin of individual HAPs was explored by examining statistical associations between personal (P) and indoor (I), and personal and outdoor (O) concentrations. Results from this evaluation are included in Table 2. In general, the analyses for both ethnic groups indicate that P and I were similar, and that P was higher than O ($p \leq 0.01$). For the majority of the studied compounds, personal and indoor concentrations were probably influenced by the same source(s), and most of the exposure occurred indoors. In a few cases, P was statistically higher than I, implying short episodic events where the participant may have been close to sources. Some exceptions to these observations included benzene and MTBE in LA, where P and O were statistically similar. Outdoor sources for these HAPs, particularly gasoline-powered vehicles, were likely dominant among participants in this city.

DISCUSSION

Few studies have examined cancer risks of minority groups from exposure to HAPs. We selected the Toxics Exposure Assessment Columbia-Harvard study (TEACH; Sax et al. 2006) for comparison purposes because, except for 1,3-butadiene, both RIOPA and TEACH considered the same compounds and unit risk factors. The TEACH study evaluated mostly participants from minority backgrounds in New York City (NYC) (African-American = 43%, Hispanic = 50%) and Los Angeles (LA_T) (Hispanic = 93%), although it only included high school students. We obtained mean (median) *CCR*s in LA, EL and HO among Hispanics of 556 (429), 962 (518) and 2,407 (699) per million, respectively. TEACH reported comparable values of 957 (666) per million in NYC and 806 (486) per million in LA_T. Sax et al. (2006) also identified formaldehyde, *p*-DCB, acetaldehyde, chloroform and benzene as the main contributors to *CCR*. *p*-DCB was

responsible for the largest discrepancies in cumulative risk between and within TEACH and RIOPA.

We compared our observations on cancer risks from exposure to *p*-DCB, chloroform, benzene and formaldehyde to those from four previous studies in the U.S.: the TEAM studies (Wallace 1991), which evaluated eight urban areas; NHANES III (Churchill et al. 2001) and 1999-2000 NHANES (D'Souza et al. 2009), which assessed the U.S. population; and NHEXAS (Clayton et al. 1999; Gordon et al. 1999), which examined six mid-western states. Both NHANES studies and NHEXAS used random and representative samples. To compare risks, we multiplied the personal concentrations reported in these investigations times the unit risk factors used in our analysis.

Our finding that Hispanics may be disproportionately affected by *p*-dichlorobenzene is supported by results from TEAM and 1999-2000 NHANES. Cancer risks from exposure to *p*-DCB for Hispanics in RIOPA (mean = 899×10^{-6}, median = 48×10^{-6}) and in particular for those who resided in Houston (mean = $1{,}782\times10^{-6}$, median = 305×10^{-6}) were significantly greater than the estimates for the general population from the TEAM studies (mean = 242×10^{-6}). Results from NHANES reinforce our observations because Hispanics from this investigation also had higher median *CR*s for *p*-DCB (52×10^{-6}) than Whites (15×10^{-6}). Common indoor sources of *p*-DCB include deodorizers/air fresheners and moth repellents (Wallace 1991). These products are often pure *p*-dichlorobenzene and are prone to relatively high mass emission rates. Answers to RIOPA questionnaires suggest that deodorizers/air fresheners are more prevalent among Hispanics than moth repellents; 59% of Hispanics reported to have used air fresheners during the study, while only 6% utilized moth repellents. Solid toilet bowl deodorants may be of particular importance as indicated by Churchill et al. (2001) whose analysis of data from NHANES III showed a two-fold positive association between recent use of this

type of product and increased blood levels of *p*-DCB. Serrano-Trespalacios et al. (2004) determined that toilet deodorants were present in 30% of the homes they monitored in Mexico City, while moth cakes were rarely found.

Chloroform also caused higher risks for Hispanics than Whites in the RIOPA and 1999-2000 NHANES studies. However, the risks we estimated for Hispanics were comparable to those from NHEXAS and TEAM for the general population. One of the problems with evaluating exposure to chloroform is that its main residential source is volatilization from chlorinated tap water, which has chloroform concentrations that are highly variable depending on the water source, date and time. Median risks for Hispanics in LA were 1.7 times higher than those estimated by TEACH in this city. This discrepancy could have been influenced by differences in behavioral patterns between the participants in RIOPA (i.e., adults) and TEACH (i.e., high school students). Nevertheless, higher personal concentrations of chloroform among Hispanics may be because these households tend to exceed the U.S. average number of people per home by a factor of 1.35 (U.S. Census Bureau 2004), which may lead to a larger than average number of showers per residence. This could contribute to increases in cancer risks, since Nuckols et al. (2005) determined that chloroform concentrations in the blood and breath are affected by emissions that occur while others are taking showers. Nuckols et al. (2005) also noted these increases in people who washed dishes by hand. Because this activity is usually performed by women, this could further explain our finding that Hispanic women had greater *CR*s for chloroform than men.

Benzene was also found to be a pollutant to which Hispanics may have higher exposures than Whites in the RIOPA and 1999-2000 NHANES studies. However, the median risk levels we estimated for Hispanics were comparable or lower than those from 1999-2000 NHANES and NHEXAS (Clayton et al. 1999) for the overall population.

This is probably because these two studies included participants who smoked while RIOPA did not, and smoking is the leading source of benzene in both personal and indoor air concentrations for the general population (Wallace 1996). Our results suggested that Hispanics may have had a high risk from exposure to benzene because of the proximity of their homes to ambient sources of HAPs. However, in Elizabeth and Houston, their personal concentrations were statistically higher than outdoor levels, and there were no statistical differences between personal and indoor concentrations. Therefore, other sources close to the living areas, such as emissions from gasoline-powered devices, could have infiltrated indoors (Batterman et al. 2007) and affected the exposure of Hispanics to benzene. The role of gasoline is supported by high median exposures of Hispanics to MTBE, a VOC emitted exclusively by gasoline.

Formaldehyde was the largest contributor to *CCR* for 69% of Hispanics and 88% of Whites. Moreover, both groups had a similar median risk for formaldehyde (276×10^{-6}). Comparable values were estimated in the TEACH study (NYC = 222×10^{-6}, LA_T = 266×10^{-6}) and the NHEXAS pilot study in Arizona (273×10^{-6}; Gordon et al. 1999). This consistency in *CR*s suggests possible uniform chronic exposures to formaldehyde throughout the U.S. population due to prevalent indoor sources such as pressed-wood materials.

Acetaldehyde was among the important contributors to cumulative cancer risk for both Hispanics and whites. The TEACH study also reported acetaldehyde to be of significance with respect to CCR. For both RIOPA and TEACH the median personal and indoor concentrations of acetaldehyde were comparable, whereas median personal concentrations were 2 to 5 times higher than outdoor concentrations. Therefore, sources within residences were as or more important than outdoor sources in terms of exposure and risk. Our evaluation suggests that combustion-related sources other than tobacco

smoke, which was excluded from the RIOPA study, may have been of relevance because personal concentrations for acetaldehyde and benzene showed statistically significant correlations (Hispanics, Spearman coefficient (r_s) = 0.22; whites, r_s = 0.32). Similar results were observed with indoor concentrations (Hispanics, r_s = 0.24; whites, r_s = 0.30). Other possible indoor sources include detergents, cleansers and liquid wax (Nazaroff and Weschler 2004).

In general, Hispanics and Whites who lived in houses with low ventilation rates had higher cancer risks from exposure to HAPs, particularly from *p*-DCB and chloroform, consistent with results from TEACH (Sax et al. 2004). The cumulative effect of AER on exposure was demonstrated by statistical differences in *CCR* between participants who lived in homes with ventilation rates below 0.5 hr^{-1} and above 1 hr^{-1}. Moreover, higher median AERs in Hispanic households in Los Angeles (1.2 hr^{-1}) than in Elizabeth (1.0 hr^{-1}) and Houston (0.5 hr^{-1}) may explain why (1) Hispanics in LA had lower *CCR*s than in the other two cities, (2) no statistical differences in *CCR* were observed in LA between Hispanics and Whites (median AER for White households = 0.8 hr^{-1}), and (3) personal and outdoor concentrations for benzene and MTBE were statistically similar for Hispanics in LA. Differences in AER among cities may be because a larger percentage of Hispanic homes in LA (74%) reported to have had their windows open for some time during the sampling session than in EL (30%) and HO (7%). Even though these results suggest that ventilation rates can reduce risks from HAPs, this measure is not sufficient. People in homes with AERs 2.9 times higher than a recommended value of 0.35 hr^{-1} (ASHRAE 2004) experienced median *CCR*s of 435×10^{-6}.

We compared our estimates with those from studies that are based on outdoor measurements. Morello-Frosch and Jesdale (2006) utilized ambient levels of HAPs from

the 1999 National Air Toxics Assessment (NATA) and reported a mean *CCR* of 632×10^{-6} for the total population in U.S. metropolitan areas, and 900×10^{-6} for Hispanics. Another investigation, the Multiple Air Toxics Exposure Study III (MATES), evaluated outdoor contamination in California's South Coast Basin. Measurements yielded a mean *CCR* of $1{,}200\times10^{-6}$ (SCAQMD 2008). Mobile sources were important in both studies, accounting for approximately 88% of the *CCR* from NATA and 94% of the *CCR* from MATES. Diesel particulate matter contributed 53% and 84% of these *CCR*s, respectively. Our analysis included a subset of the HAPs that were evaluated in the other two studies and these contaminants were predominantly of indoor origin. Nevertheless, our estimates of mean *CCR*s for Hispanics ranged from 556×10^{-6} to $2{,}407\times10^{-6}$ and were either comparable or greater than those from NATA and MATES.

Although the cancer risk assessment that we performed was a useful tool to place into context the measured personal concentrations from RIOPA in a standardized manner, the approach has limitations. Our calculations underestimate cumulative risk because we only analyzed 12 HAPs. Important contributors to cancer risk that were not part of our evaluation are polycyclic organic matter (POM) and 1,3-butadiene, for which Woodruff et al. (2000) estimated *CR*s of 72×10^{-6} and 31×10^{-6}, respectively, using outdoor measurements. Other limitations include uncertainty in the derivation of cancer potency factors. Furthermore, cancer potencies assume 70-year lifetime exposures. Our estimates, like those of others reported herein, are based on a sample of this exposure. Additionally, the results of our evaluation should be considered with caution because the RIOPA participants were not selected using a random, stratified sampling scheme. Finally, the statistically significant discrepancies in *CCR* that we report between Hispanics and Whites are primarily based on measurements from Elizabeth and Houston. As explained earlier, disparities were not observed in Los Angeles, likely because higher

ventilation rates mitigated the effect of indoor sources. Despite these limitations, our analysis together with results from prior studies appear to provide compelling evidence for the assumption that air pollutant-related cancer risk disparities between Hispanics and non-Hispanic Whites are indeed likely, and substantiate the importance of the contribution from indoor air pollution to these risks.

CONCLUSIONS

Median cumulative cancer risk (*CCR*) for Hispanics and Whites were two orders of magnitude greater than the EPA benchmark of 10^{-6}. Risk estimates among the top 10^{th} percentile of Hispanics were greater than 10^{-3}. *CCR* for both ethnic groups was dominated by five of the 12 HAPs included in the study: formaldehyde, *p*-DCB, acetaldehyde, chloroform and benzene. Exposure to all of these compounds but benzene was primarily dominated by indoor residential sources. Formaldehyde was the largest contributor to *CCR* for 69% of Hispanics and 88% of Whites. Hispanics had higher exposures to some of these pollutants, leading them to have statistically higher *CCR* estimates than Whites. This outcome was mainly due to *p*-DCB, probably associated with the use of air fresheners that emit this VOC. Increases in house ventilation rate can decrease risks. However, our findings suggest that strategies to lower exposure to HAPs among groups that are at greater risk, as well as for the general population, should consider both improved ventilation and concurrent reductions in indoor sources of the HAPs included in this study.

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APPENDIX B

Formaldehyde in Residences:
Long-Term Indoor Concentrations and Influencing Factors

(Accepted to *Indoor Air*)

ABSTRACT

Chronic human exposure to formaldehyde is significantly increased by indoor sources. However, information is lacking on why these exposures appear to persist in older homes with aging sources. We use data from the Relationships of Indoor, Outdoor, and Personal Air (RIOPA) study to evaluate 179 residences, most of which were older than 5 years. We assess the dependence of indoor formaldehyde concentrations (C_{in}) on building type and age, whole-house air exchange rate, indoor temperature, and seasonal changes. Indoor formaldehyde had mean and median concentrations of 17 ppb, and primarily originated from indoor sources. The factors we analyzed did not explain much of the variance in C_{in}, probably because of their limited influence on mechanisms that control the long-term release of formaldehyde from aging pressed-wood products bound with urea-formaldehyde (UF) resins. We confirmed that the mitigating effects of ventilation on C_{in} decrease with time through the analysis of data for new homes available in the literature, and through models. We also explored source control strategies and conclude that source removal is the most effective way to decrease chronic exposures to formaldehyde in existing homes. For new homes, reducing indoor sources and using pressed-wood with lower UF content are likely the best solutions.

PRACTICAL IMPLICATIONS

Formaldehyde concentrations in homes due to indoor sources appear to persist throughout the lifetime of residences. Increases in ventilation rates are most effective in decreasing indoor concentrations in new homes where formaldehyde levels are high or

when homes are tight. Consequently, other alternatives need to be promoted such as decreasing the amount of pressed-wood products with urea-formaldehyde (UF) resins in homes or reducing the UF content in these materials.

KEYWORDS

Formaldehyde, air exchange rate, pressed-wood products, emissions, RIOPA.

INTRODUCTION

Formaldehyde is a hazardous air pollutant that is widespread in residential buildings. It is a potent irritant and is classified as a probable human carcinogen (Group B1) by the U.S. Environmental Protection Agency (EPA). Hun et al. (2009) and Sax et al. (2006) concluded that formaldehyde was the highest contributor to the cumulative cancer risk from exposure to air contaminants that are typically found in residences. The state of California set 9 $\mu g/m^3$ (7 ppb at 25 °C) as the chronic Reference Exposure Level (REL) for formaldehyde (Office of Environmental Health Hazard Assessment; OEHHA, 2008). The REL is based on positive associations, especially among children with diagnosed asthma, between prolonged exposures to formaldehyde and allergic sensitization, respiratory symptoms (e.g., coughing, wheezing), or decrements in lung function (OEHHA, 2008). However, Gordon et al. (1999), Sax et al. (2006) and Weisel et al. (2005) monitored homes across the U.S. that are representative of the housing stock and reported median residential indoor concentrations of formaldehyde (C_{in}) that are between 1.9 and 2.4 times higher than the REL. These investigators also reported C_{in} to be greater than outdoor concentrations, which is indicative of the presence of indoor sources. There are numerous sources of formaldehyde indoors, such as consumer products (Gupta et al., 1982; Kelly et al., 1999) and chemical reactions with ozone (Morrison and Nazaroff, 2002; Weschler, 2000). However, pressed-wood products (PWP) with UF resins (i.e., particleboard, medium-density fiberboard (MDF) and

hardwood plywood) are major sources because they have high formaldehyde emission rates and are widely used in residences for cabinetry, furniture, and house construction (Gupta et al., 1982; Kelly et al., 1999; Mølhave et al., 1995). The use of PWP in homes is not expected to decline in the future; consequently, California recently enacted regulations that will reduce formaldehyde emissions from these materials to lessen exposure to formaldehyde (California Air Resources Board; CARB, 2007).

Numerous product-specific factors influence formaldehyde emission rates from PWP. Meyer and Hermanns (1985a) noted that material structure and porosity can affect the release rate of formaldehyde. Myers (1984a) determined that materials with resins that had a higher formaldehyde-to-urea mole ratio tended to have higher emission rates. Myers (1984a) also reported that longer press time and/or higher temperature during manufacturing of pressed-wood products can lower emissions. Additionally, the release of formaldehyde from PWP is decreased by overlaying barriers such as paint coatings, vinyl laminate and carpet (Haneto, 1986; Hodgson et al., 2002; Kelly et al., 1999).

Once pressed-wood products are in use, numerous building properties can affect indoor formaldehyde concentrations. In general, C_{in} is positively correlated with source emission rate and loading factor, defined as the ratio of the emitting surface area and space volume; and negatively associated with air exchange rate (AER) (Myers, 1984b). High emission rates are expected when materials are new and contain high concentrations of formaldehyde, but emissions usually decrease as buildings and materials age (Dingle and Franklin, 2002; Stock and Mendez, 1985). Furthermore, emission rates rise with increases in indoor temperature, relative humidity (*RH*), or air velocity over the emitting surface, as well as with decreases in C_{in} (Matthews et al., 1984; Myers, 1985; Silberstein et al., 1988). Other influential processes include adsorption and desorption of formaldehyde to and from material surfaces typically found in buildings such as gypsum

board (Matthews et al., 1987). Adsorption and desorption processes tend to decrease the decay rate of C_{in}.

Despite the large number of factors that can affect indoor formaldehyde concentrations and emission rates from PWP, relatively comparable C_{in} values have been observed in existing residences (i.e., > 1 year old) in the United States. The National Human Exposure Assessment Survey (NHEXAS) pilot study in Arizona assessed 189 homes and found a median formaldehyde concentration of 17 ppb (Gordon et al., 1999). The Relationships of Indoor, Outdoor, and Personal Air (RIOPA) study included 311 residences in three U.S. urban areas (Weisel et al., 2005). A median C_{in} of 16 ppb was observed. The Toxics Exposure Assessment Columbia-Harvard (TEACH) study involved 46 homes in New York City and 41 residences in Los Angeles; median C_{in} were 13 and 15 ppb, respectively (Sax et al., 2006). These investigations indicate that the U.S. population may be experiencing chronic and comparable exposures to formaldehyde. These exposures may be of significance among children as positive associations between formaldehyde concentrations that are typically found in homes (median = 13 ppb) and atopy have been reported (Garrett et al., 1999).

The objective of this study is to examine reasons behind the apparent chronic and similar exposure to formaldehyde in the United States. We analyze data from homes that participated in the RIOPA study, and evaluate the effect of factors that have been commonly assumed to influence C_{in} such as building age and type, house AER, indoor temperature, and seasonal changes. Furthermore, we attempt to explain the limited effect of these factors on C_{in} by investigating how these relationships are influenced by the release mechanisms of formaldehyde from aging PWP. In addition, we explore strategies to decrease indoor formaldehyde concentrations such as increases in ventilation rate, source control, and air cleaning technologies.

METHODOLOGY

This research is based on the analysis of data from a sample of houses without resident smokers that participated in the RIOPA study. Data were made available by the Health Effects Institute (HEI, 2008). Approximately 100 residences volunteered in each of Los Angeles County, California, Elizabeth, New Jersey, and Houston, Texas. Participants in Houston and Elizabeth constitute a convenience sample, while the participants from Los Angeles were a subset from a randomly selected sample of individuals from a previous study.

Weisel et al. (2005) provide a detailed description of the RIOPA field and measurement protocols. From 1999 to 2001, homes were monitored during two 48-h periods that were approximately 3 months apart. Air samples were collected concurrently inside and outside each home. Formaldehyde was measured using the Passive Aldehydes and Ketones Sampler (PAKS) coupled with HPLC-fluorescence analysis (Zhang et al., 2001). Concentrations at or below the respective method detection limit (MDL) were censored by replacement with ½ the MDL concentrations. In addition to monitoring the air, building characteristics and daily indoor/outdoor personal and household activity patterns were collected during each of the sampling sessions by means of questionnaires and walkthrough surveys. AER was concurrently measured using a perfluorocarbon tracer (PFT) method. Temperature and *RH* were recorded every 5 min using a HOBO sensor (HOBO, Onset Computer Corp, Bourne, MA, USA). Temperature, *RH* and AER were reported as time-average values for the sampling period.

Households where someone smoked during a sampling period were excluded from this assessment. Ventilation rates greater than 5 h^{-1} were also excluded because the PFT method is unreliable at these values. Residences where volumes were recorded to be < 80 m^3 were not included in the analysis because it is highly probable that these values

were not correct. Data from the first visit were selected when information on the building age or type from the first and second sampling sessions differed. Only data from homes with values for indoor and outdoor concentrations, AER, and indoor temperature were employed. Averages were calculated when all of these measurements were available for the two monitoring sessions because these are dependent variables that describe a single household. These provisions reduced the overall sample size from 311 to 179.

Nonparametric statistical analyses were utilized because the data were generally positively skewed. Associations between variables were evaluated with Spearman rank-correlation coefficients (r_s). Correlation coefficients were also used with C_{in} and temperature because their relationship is approximately linear for temperatures between 20 and 30 °C. The Wilcoxon sign-rank test was used to assess differences between paired samples, such as concurrent indoor and outdoor concentrations. The Wilcoxon rank-sum test was utilized to evaluate differences between two independent samples, such as indoor concentrations from single-family detached homes and apartments. Similarly, the Kruskal-Wallis test was used with three or more levels. Differences were considered statistically significant at $p \leq 0.05$. SPSS version 15.0 (SPSS Inc., IBM, Chicago, IL, USA) was employed for these analyses.

RESULTS

The largest number of homes was located in Los Angeles (n = 73), followed by Elizabeth (n = 58) and Houston (n = 48). The majority of these residences were either single-family detached homes (50%) or apartments (37%). Of the 179 homes, 23 were less than 5 years old, of which 21 were apartments in Los Angeles. Ten out of 12 manufactured homes were older than 5 years; building age was not available for one of these houses. Fifty-eight percent of the households that were monitored once (n = 78)

were visited during the summer, while 51% of the residences that were sampled twice (n = 101) had their 1st and 2nd visits during summer and fall, respectively.

Formaldehyde concentrations and building measurements

Table 1 presents summary statistics for formaldehyde concentrations and other building-related measures. More than 99% of the formaldehyde concentrations were above the method detection limit of 0.14-1.0 ppb. Indoor concentrations of formaldehyde (C_{in}) followed approximately a normal distribution as is depicted by its cumulative distribution curve in Figure 1a. Both the mean and median C_{in} were approximately 17 ppb, and a relatively small coefficient of variation (CV) of 26% was observed. Mean and median outdoor formaldehyde concentrations (C_{out}) were about 32% of C_{in}. Indoor temperatures (T) had minimal variation (mean and median = 24 °C, CV = 7%), probably because of moderate weather, or use of air conditioners or heaters during the monitoring session. The mean (median) percent of the sampling period in which windows were reported to have been open in homes in Los Angeles and Elizabeth were 50% (50%) and 35% (31%), respectively. The mean (median) percent of the monitoring session when the air was reported to have been conditioned in homes in Los Angeles and Elizabeth were 9.0% (0%) and 7.9% (0%), respectively. Conversely, residences in Houston had open windows during shorter portions of the study (mean = 7.2, median = 0%) and instead conditioned the air during a significant part of the sampling period (mean = 69, median = 96%). Window openings likely contributed to lower air AERs for Houston homes (mean = 0.59, median = 0.44 h^{-1}) relative to homes in Elizabeth (mean = 1.2, median = 0.94 h^{-1}) and Los Angeles (mean = 1.3, median = 0.92 h^{-1}), as well as to a high variability in the AER for all three cities (mean = 1.1, median = 0.80 h^{-1}, CV = 77%). Indoor RH values were only available for 59 of the 179 homes because they were not reported in Los

Angeles and because of equipment malfunction. Mean and median *RH* was 44% with a CV of 27%.

Table 1. Descriptive summary of measurements.

Measurements	*n*	Mean	SD	Median	5th %tile	95th %tile	CV (%)
Indoor concentration (C_{in}, ppb)	179	17.2	4.49	16.8	10.1	24.8	26
$C_{in,\,std}$ [a] (ppb)	179	20.2	7.72	19.8	10.1	32.7	38
Outdoor concentration (C_{out}, ppb)	179	5.51	3.30	5.43	2.36	7.65	60
$C_{in} - C_{out}$ (ppb)	175	12.2	4.80	11.4	5.00	20.0	39
Volume (m^3)	179	232	144	200	92.0	468	62
Air exchange rate (AER, h^{-1})	179	1.07	0.82	0.80	0.20	2.83	77
Temperature (*T*, °C)	179	24.1	2.03	24.0	20.9	27.4	8.5
Relative humidity (*RH*, %)	59	44.5	12.2	45.0	19.0	63.5	27

[a]Estimated with equation from Meyer and Hermanns (1985b).
[b]Four cases where $C_{out} > C_{in}$ were excluded.
Abbreviations: CV, coefficient of variation; SD, standard deviation.

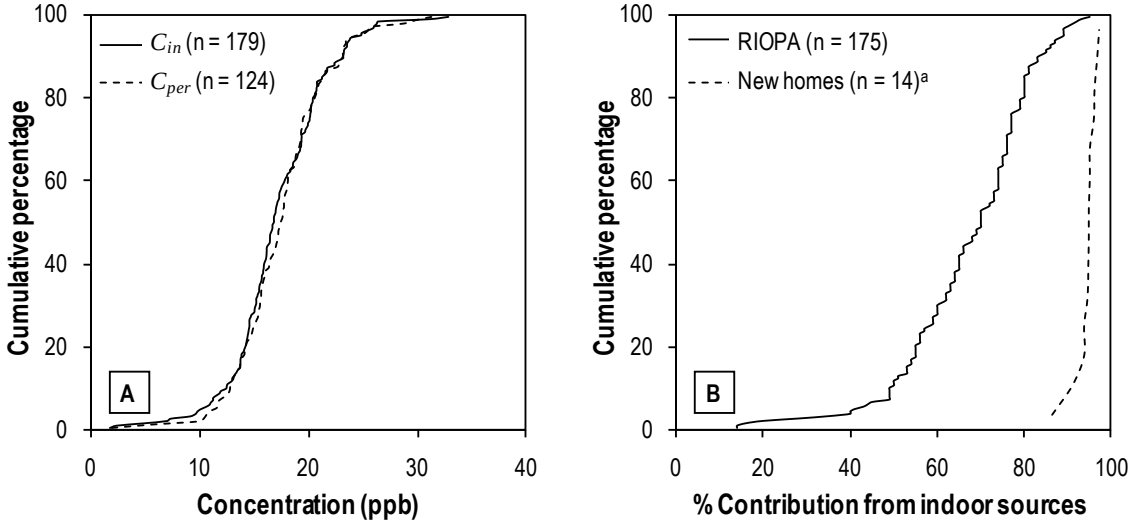

Figure 1. Cumulative distribution functions for indoor (C_{in}) and personal (C_{per}) formaldehyde concentrations (A) and the percent contribution from indoor sources (B).
[a]Data from Hodgson et al. (2000) and Sherman and Hodgson (2004).

Indoor and outdoor formaldehyde sources

Measurements of C_{in} were statistically higher than C_{out} ($p \leq 0.01$). The percent contribution of indoor sources to indoor concentrations (I_{cont}) was calculated by subtracting C_{out} from C_{in} and dividing the result by C_{in}. Four homes where C_{out} was higher than C_{in} were excluded from the analysis. Figure 1b shows the cumulative distribution curve for I_{cont} and indicates that in half of the RIOPA houses more than 70% of C_{in} originated from indoor sources. Additionally, the contribution of indoor sources to C_{in} was greater than that from outdoor sources for 90% of the homes. Figure 1b also presents I_{cont} estimated for new homes from data published by Sherman and Hodgson (2004). Their investigation involved seven manufactured and seven conventional homes less than 1 year old. The value of C_{out} was estimated to be 2 ppb based on a related study by Hodgson et al. (2000). Figure 1b shows that indoor sources accounted for at least 95% of C_{in} in 50% of new homes.

Influencing factors on indoor formaldehyde

No correlation was observed between C_{in} and T (n = 179) or RH (n = 59). Seasonal effects were evaluated by examining formaldehyde concentrations from residences that were monitored twice. Only the 10 homes that were visited in the fall and winter (median: $C_{in,\,fall}$ = 20 ppb, $C_{in,\,winter}$ = 15 ppb, $C_{in,\,\mathrm{fall}} - C_{in,\,\mathrm{winter}}$ = 5.8 ppb) showed differences in concentration that were statistically significant. RH was the only other variable that was statistically different between these two seasons (median: RH_{fall} - RH_{winter} = 22%), but RH values were only available for six of the residences, most of which had differences in C_{in} that were in the upper 50th percentile. Inferences on the relationship between concurrent measurements of C_{in} and RH should be drawn with caution because it can take days or weeks for formaldehyde emissions from composite wood to reach equilibrium after changes in humidity (Myers, 1985). Homes that were

visited twice during other seasons showed statistically significant differences in AER and/or *T*, although these were not accompanied by statistically significant differences in C_{in}.

Indoor formaldehyde concentration was not statistically correlated with AER, building type or age. Homes with ventilation rates in the lower and upper 50th percentile both had a median C_{in} of 17 ppb. Manufactured homes had a median C_{in} value (18 ppb, n = 12) that was slightly higher than that for apartments (16 ppb, n = 67) and single-family detached homes (17 ppb, n = 89). For apartments, the reported AER include infiltration through walls shared with neighbors; consequently, C_{in} is likely to be affected by formaldehyde concentrations in adjacent dwellings. Building age was not an important determinant; there was minimal difference in median concentration (0.1 ppb) between homes newer (n = 23) and older (n = 124) than 5 years. However, note that 21 of the homes less than 5 years old were apartments in Los Angeles.

The effects of AER and building type were assessed concurrently by dividing the infiltration rates of single-family detached homes and apartments into three categories (< 0.62, 0.62 – 1.17, > 1.17 h^{-1}). These ranges, and those to follow, were selected to minimize differences in sample size among AER categories. The formaldehyde concentration for each of these scenarios is depicted in Figure 2a. Differences in C_{in} between building types within each of the ventilation ranges, and among AER categories within building types, were not statistically significant. Similarly, to evaluate the effect of AER and building age, ventilation rates were grouped into two ranges ($\leq$ 0.75, > 0.75 h^{-1}) and the age of the residence was divided into three categories (< 5, 5 – 40, > 40 years old) as shown in Figure 2b. No statistically significant differences in C_{in} were observed due to building age within each of the ventilation ranges. For homes older than 40 years,

C_{in} was statistically higher when ventilation rates were ≤ 0.75 h^{-1} than otherwise; the median C_{in} for these two scenarios differed by 1.3 ppb.

The aforementioned assessments were repeated with formaldehyde concentrations that were standardized to 25 °C (Meyer and Hermanns, 1985b) to control the effects of temperature. The analyses were also performed with (C_{in} – C_{out}) to account for background concentrations. In general, results from these evaluations were similar to those obtained with C_{in}.

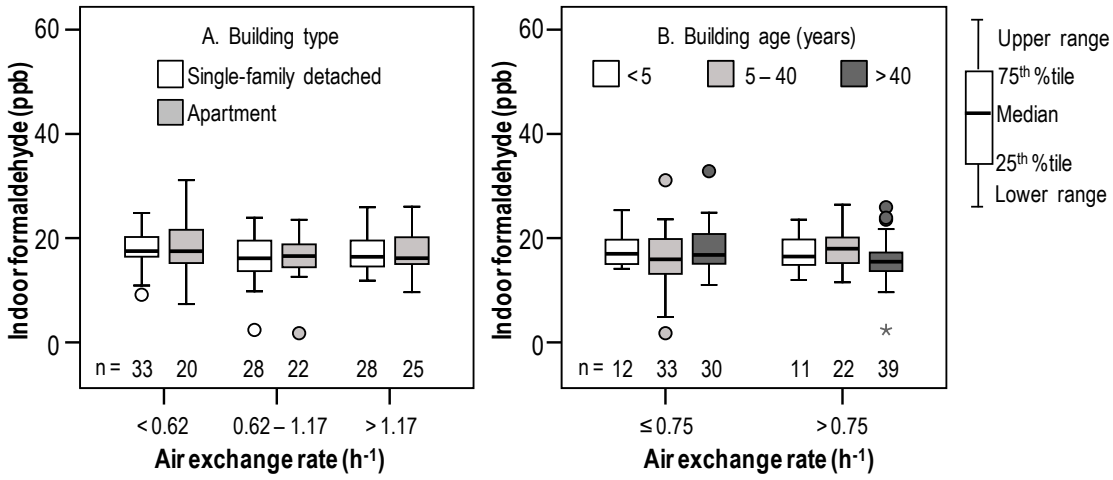

Figure 2. Indoor concentrations of formaldehyde for various air exchange rate categories and building type (*A*) and building age (*B*). 'o' and '⋆' are values between 1.5 and 3, and more than 3 times the interquartile range, respectively, from the 25th or 75th percentiles.

DISCUSSION

Results from the RIOPA study and from other exposure assessments (Dingle and Franklin, 2002; Gordon et al., 1999; Sax et al., 2006) suggest that indoor formaldehyde concentrations are ubiquitous in residences of all ages. Pressed-wood products (PWP) are likely the most significant contributors to C_{in} because they are among the highest emitters of formaldehyde (Gupta et al., 1982; Kelly et al., 1999) and considerable

amounts of these materials are generally present in homes. Dingle and Franklin (2002), Stock and Mendez (1985) and Versar Inc. (1986) examined indoor formaldehyde concentrations in homes that were 0 to 100 years old, and developed models to estimate C_{in} with respect to building age (R^2 = 0.19 to 0.42). All of these models predict decreases in C_{in} with time that concur with the mechanisms that affect the release of formaldehyde from PWP bound with UF resins (Gammage and Gupta, 1984; Nelms et al., 1986). The first mechanism is responsible for the high emission rates from new materials, and involves mostly the evaporation of free formaldehyde in UF resins as well as the breakdown of easily hydrolyzed chemical bonds. Emissions of free formaldehyde are significantly affected by temperature and *RH*. As these sources are depleted, the second mechanism becomes dominant and formaldehyde is generated from further hydrolysis of the polymeric structure of UF resins. This mechanism is long-lasting, involves low emission rates, and is likely to be controlled by diffusion from within the material to its surface. The second release mechanism was likely prevalent in the existing homes we evaluated because it characterizes the long-term formaldehyde concentrations we observed and their minimal dependence on temperature given the low availability of free formaldehyde in older materials.

Personal concentrations of formaldehyde (C_{per}, n = 124) were also measured in the breathing zone of participants throughout their daily activities during the RIOPA study. Our analysis indicates that C_{per} and C_{in} were statistically similar, and that their cumulative distribution curves were almost identical as shown in Figure 1a. This suggests that exposures were highly related to indoor residential sources. The median C_{per} of 17 ppb exceeded California's REL of 7 ppb (OEHHA, 2008). We assessed various methods to decrease C_{in} since it is so strongly correlated with actual exposures to

formaldehyde. We examined mitigation strategies such as increase in ventilation rate, source reduction, and air cleaning technologies.

Previous studies indicate that increases in AER can lower C_{in}, but these studies involved chamber tests with new pressed-wood materials (Myers, 1984b) or tightly-built homes with mean ventilation rates of 0.2 h^{-1} (Gilbert et al., 2006). Measurements from the RIOPA study indicate no association between ventilation and C_{in}, perhaps because the mitigating effects of AER are influenced by material aging and building tightness as suggested by Meyer and Hermanns (1985a). This lack of association is consistent with the theoretical rate of change of C_{in} with respect to AER (Equation 1). We obtained this equation by combining a steady-state equation for indoor concentration assuming a well-mixed house and an emission rate equation that is based on mass transfer away from a source for which an equilibrium concentration can be defined at the source-air interface.

$$\frac{dC_{in}}{d\lambda} = \frac{-k_g L(C_{eq} - C_{out})}{(\lambda + k_g L)^2} \qquad [1]$$

Here, C_{out} is the outdoor concentration (ppb), L is the loading factor (m^2/m^3) and denotes the ratio of surface area and space volume, λ is the AER (h^{-1}), k_g is the mass transfer coefficient (m/h), and C_{eq} is the equilibrium concentration at the surface of the emitting material (ppb). Equation 1 shows that C_{in} should decrease with increases in AER (λ), but $|dC_{in}/d\lambda|$ may diminish as materials age and C_{eq} declines, or in homes with high AERs. $|dC_{in}/d\lambda|$ may also decrease at higher ventilation rates because k_g tends to be positively associated with AER.

We estimated $dC_{in}/d\lambda$ for tightly-built new houses (mean C_{in} = 42 ppb, mean AER = 0.4 h^{-1}, n = 14) based on information from Sherman and Hodgson (2004), and an outdoor concentration of 2 ppb as per Hodgson et al. (2000). We performed the same

calculations for existing residences (mean C_{in} = 17 ppb, mean AER = 1.7 h^{-1}, n = 179) with data from the RIOPA study. Results from linear regressions indicate that $dC_{in}/d\lambda$ values for tightly-built new homes (-45.7 ppb×h, p = 0.10) were two orders of magnitude greater than those for existing residences (-0.56 ppb×h, p = 0.20). Additionally, C_{in} showed a higher dependence on AER in new homes (R^2 = 0.2) than in older homes (R^2 = 0.01).

Source control strategies to reduce C_{in} include decreasing the amount of PWP in homes, utilizing emission barriers on PWP, and lowering the formaldehyde content in PWP. Hardwood plywood paneling used to constitute about 62% of the PWP in manufactured homes, and approximately 14% in conventional homes (Versar Inc., 1986). In modern homes, interior plywood wall paneling has been replaced with gypsum board in manufactured homes so that new manufactured and site-built homes constructed in the late 1990s had comparable C_{in} (Hodgson et al., 2000). Furthermore, we noted similar median indoor concentrations in manufactured homes (18 ppb) and single-family detached homes (17 ppb). Another source control strategy involves emission barriers on PWP. Haneto (1986) estimated that the emission factor (*EF*), or the emission rate per unit surface area, from particleboard may be lowered by 19% with a latex paint coating and 94% with an alkyd paint coating. Hodgson et al. (2002) noted that *EF* from a standard passage door was 31 times higher than that from a similar door covered with vinyl sheets. This ratio was 1.3 for emissions from plywood underlayment without and with residential carpet and standard bonded polyurethane cushion (Hodgson et al., 2002).

We examined possible effects from the California standards that reduce formaldehyde content in pressed-wood materials (CARB, 2007). Industry average concentrations in 2002 were 90, 180 and 250 ppb for hardwood plywood, particleboard and medium-density fiberboard (MDF), respectively, based on ASTM Standard Test

Method E 1333 (2002) and CARB (2007). By 2012, formaldehyde concentrations from these products should not exceed 50, 90, and 110 ppb, respectively. We calculated *EF* per E 1333 (hardwood plywood = 58, particleboard = 105, MDF = 128 ppb×m/h), and estimated C_{in} from PWP with Equation (2).

$$C_{in} = \frac{\left(\sum_{i}^{n} EF_i \times L_i \times (1 - RF_i) \right)}{\lambda} \quad [4]$$

where, *RF* is an emission reduction factor due to barriers, and *i* accounts for various composite products and their use. Table 2 summarizes the loading factors and *RF* values in a 186 m^2 single-family detached home. We did not include furniture because we did not find their loading factors in the literature; however, furniture is a very important source given that it is more easily and commonly reintroduced in homes. Using a median AER of 0.25 h^{-1} for new California residences (Offermann et al., 2008) and 95% of the house volume, we estimated C_{in} to be 73 ppb. Although our estimates are high-end values that will diminish with time, it is not evident that C_{in} will meet the REL of 7 ppb (OEHHA, 2008), because our calculations did not include furniture made with PWP and various consumer goods that emit formaldehyde. Nonetheless, we estimated a considerably higher C_{in} of 140 ppb using the 2002 industry average concentrations for PWP, indicating a dramatic reduction due to regulations in California.

Other alternatives to decrease indoor formaldehyde concentrations include air cleaning technologies with adsorbent materials. Sekine and Nishimura (2001) developed an air cleaner for formaldehyde using activated carbon and manganese oxide. The device was tested in a new multi-family home for 7 months; C_{in} decreased from 210 to 40 ppb. These results imply that increases in ventilation rates to reduce C_{in} in new tightly-built homes could be moderated with the use of air cleaners, which may diminish the energy

consumed to decrease C_{in}. We found no information on the performance of air cleaners in existing residences with low C_{in}. More research is needed to assess the effectiveness of air cleaning technologies.

Table 2. Estimates for loading factors and emission reduction factors in single-family detached homes.

Use	L (m^2/m^3)[a]	Materials[a]	EF (ppb×m/h)	RF (%)	C_{in} (ppb)
Baseboards	0.015	MDF[a]	128	19[b]	6.4
Cabinets	0.034	Hardwood plywood[a]	58	48[c]	4.1
	0.112	Particleboard[a]	105	48[c]	24
	0.059	MDF[a]	128	48[c]	16
Countertops	0.016	Particleboard[a]	105	48[c]	3.5
Doors	0.049	Hardwood plywood	58	19[b]	9.3
Entertainment center	0.044	Hardwood plywood[a]	58	97[d]	0.3
Shelving	0.034	Hardwood plywood[a]	58	19[b]	6.3
	0.009	Particleboard[a]	105	19[b]	3.1
Total	**0.372**				**73**

[a]Loading factors and materials for a 186 m^2 single-family detached home (CARB, 2007). These estimates do not include furniture.
[b]*RF* for latex paint (Haneto, 1986).
[c]*RF* for vinyl laminate on one side of the material (Hodgson et al., 2002).
[d]*RF* for vinyl laminate on both sides of the material (Hodgson et al., 2002).
Abbreviations: C_{in}, indoor concentration; *EF*, emission factor; *L*, loading factor; MDF, medium-density fiberboard; *RF*, emission reduction factor.

Although our assessment of the RIOPA dataset provides compelling evidence that existing homes have persistent indoor formaldehyde concentrations that are not significantly affected by factors that have been deemed as influential, some limitations should be considered. First, our results may have differed if we had analyzed data from a longitudinal study instead of a cross-sectional evaluation. However, Hawthorne et al. (1986) performed a comprehensive longitudinal investigation of formaldehyde in residences, and also noted that the degree of influence of various factors on C_{in} decreased as homes aged. Second, we did not perform a thorough assessment of all indoor sources because data from RIOPA did not allow for such an evaluation, and instead assumed that

formaldehyde originated primarily from pressed-wood materials given their widespread use in houses and long-lasting emissions. With regards to ozone-related chemistry that can introduce formaldehyde indoors, we performed a short examination with the Houston homes. We obtained 1-h maximum outdoor ozone concentrations from the Texas Commission on Environmental Quality (TCEQ, 2009), and selected data based on the sampling date and the monitoring station that was closest to the residence. Our analysis was similar to the one performed by Loh et al. (2008), but our conclusions differed in that we did not observe statistical associations between C_{in} and ozone levels. These results may have been partly influenced by the use of air conditioning systems in the Houston homes during a significant part of the sampling period (mean = 69%, median = 96%). This would limit ozone infiltration to occur through cracks in the building envelope, which may increase the loss of ozone due to chemical reactions with building envelope materials. Zhang et al. (1994) also reported poor correlations between C_{in} and indoor ozone concentrations due to strong emissions from other formaldehyde sources. Consequently, ozone-related chemical reactions may not be a major contributor of formaldehyde indoors.

CONCLUSION

Our assessment of the RIOPA database, together with results from prior studies, suggests that sources within residences are chronically exposing the U.S. population to formaldehyde. Our results suggest that median personal and indoor concentrations of 17 ppb exceed California's chronic REL by a factor of 2.4. Pressed-wood materials with UF resins are likely to be the dominant source indoors. Factors that have been identified as influential on formaldehyde concentrations due to emissions from new composite wood products did not explain much of the variance we observed in a sample of existing residences. The long-lasting release of formaldehyde from aging PWP may be controlled

by slow diffusion within the material, which may not be highly affected by the factors we examined such as house ventilation rate. Thus, reducing long-term exposures to formaldehyde in existing homes may require removing some of the sources. In houses that are to be built, source control strategies could include the use of composite wood materials with lower UF content such as those being promoted in California.

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APPENDIX C

Indoor Residential Concentrations of BTEX and MTBE

(Submitted to *Building and Environment)*

ABSTRACT

Attached garages have been identified as important sources of indoor residential air pollution. However, the literature lacks information on (1) how the proximity of cars to the living area affect indoor concentrations of gasoline-related compounds, such as benzene; (2) the origin of these pollutants, i.e., vapor or tailpipe emissions; and (3) the effect of ventilation rates on indoor concentrations of these contaminants. We analyzed data from the Relationships of Indoor, Outdoor, and Personal Air (RIOPA) study to evaluate indoor (C_{in}) and outdoor (C_{out}) concentrations for 114 residences with cars either in an attached garage, a detached garage or carport, or without cars. Results indicate that single-family detached homes with cars in attached garages were affected the most by parked vehicles, followed by homes with vehicles in carports. Concentrations in homes with cars in detached garages were similar to those in residences without cars. Low ventilation rates exacerbated C_{in} in homes with attached garages. In general, the contribution from gasoline-related sources to indoor benzene and MTBE concentrations appeared to have been dominated by car exhaust, or by a combination of tailpipe and gasoline vapor emissions. Residing in a home with an attached garage could lead to benzene exposure estimates that are an order of magnitude higher than exposures from commuting in a car in heavy traffic, and with a mean cancer risk of 17×10^{-6}. Strategies to lower exposure to gasoline-related contaminants in homes include improving construction practices to prevent the infiltration of pollutants into the living quarters or incorporating detached garages.

KEYWORDS

Attached garages, benzene, exposure, gasoline, RIOPA

INTRODUCTION

Various gasoline-related volatile organic compounds (VOCs) have been identified by the U.S. Environmental Protection Agency (EPA) as hazardous air pollutants. Benzene, toluene, ethylbenzene and xylenes (BTEX) vaporize from liquid gasoline, and are emitted in car exhaust and by some consumer products. Methyl tert-butyl ether (MTBE) was emitted almost entirely by gasoline, making it an ideal tracer for gasoline-related exposures. MTBE has been used as an additive in reformulated gasoline to comply with the 1990 Amendments to the Clean Air Act on minimum oxygen content requirements. Between 2000 and 2009, 25 states in the U.S. banned MTBE because it contaminates groundwater through leaks in gasoline pipelines and underground storage tanks, as well as surface waters by deposition from ambient air to reservoirs. Due to its high solubility in water and relatively low Henry's law constant [1], indoor exposure to MTBE in residential water supplies is likely dominated by ingestion instead of inhalation. In 2006 the U.S. EPA removed the oxygen content requirement in reformulated gasoline [2]; therefore, the use of MTBE is no longer widespread in gasoline and it can no longer serve as a gasoline tracer.

Benzene has been classified by the U.S. EPA as a known human carcinogen (Group A), and risk assessments among nonsmoking populations have repeatedly identified benzene as an important contributor to cumulative environmental cancer risk [3-6]. Elevated concentrations of ethylbenzene and MTBE have also been related to the formation of tumors in non-human animals [7]. Chronic exposure to high concentrations of toluene has been associated with neurological effects, and continuous contact with elevated concentrations of xylene isomers can impair motor coordination [8].

Investigations have found that BTEX and MTBE concentrations inside homes are exacerbated by sources within attached garages. Dodson et al. [9] determined that houses with garages had statistically higher indoor concentrations for BTEX and MTBE than residences without this source of pollution. Batterman et al. [10] and Thomas et al. [11] reported that BTEX levels were five to 18 times higher in garages than in the adjacent living area of single-family homes. Thomas et al. [11] concluded that garages can introduce similar or higher amounts of benzene indoors as tobacco smoke. Mass transfer rates of benzene from the garage to the living area were estimated to range from 24 to 26,000 μg/h in three homes, whereas a home with three smokers had indoor source strengths for benzene that varied from 150 to 13,000 μg/h. Sources of BTEX in garages include gasoline-powered devices and consumer products such as paints, cleaners, detergents, adhesives, paint thinners and oils/lubricants [12-14].

The migration of VOCs to the occupied space is due in part to the fact that the shared wall between the garage and the living quarters tends to be among the leakiest components of the house envelope [15], and to the presence of heating, ventilation and air conditioning (HVAC) components in some attached garages [16]. Batterman et al. [10] estimated that about 6.5% of the whole-house air exchange rate can originate from the attached garage. The contribution of sources within garages to indoor concentrations of BTEX and MTBE has been determined to range from 9 to 85% [9, 10, 17]. Even though exposure to BTEX commonly occurs in many microenvironments, personal concentrations for these compounds have been primarily associated with attached garages [18, 19] since Americans spend on average nearly 70% of their time in their homes [20].

Although previous work provides compelling evidence that pollutants in attached garages can infiltrate and influence indoor residential environments, this is only one of several locations where vehicles are parked at or near homes. The literature lacks

information on how other scenarios that vary the proximity of cars to the living area affect indoor concentrations of BTEX and MTBE. Among the studies that have evaluated attached garages, only Dodson et al. [9] monitored MTBE, an ideal tracer for gasoline in U.S. states where it has not been banned. However, their analysis did not distinguish among the various gasoline-powered devices found in homes, and the actual presence of cars in attached garages or in any other locations adjacent to residences.

In this study we examine how the proximity of parked vehicles next to living quarters affects indoor concentrations of BTEX and MTBE. To this end, we analyze data from nonsmoking homes that participated in the Relationships of Indoor, Outdoor, and Personal Air (RIOPA) study. In particular, we evaluate indoor and outdoor concentrations for six scenarios: single-family detached (SFD) homes with cars in the (1) attached garage, (2) detached garage, or (3) adjacent carport; (4) manufactured homes with cars in adjacent carports; (5) SFD homes with attached garages but no cars; and (6) SFD homes without both attached garages and cars. We use MTBE measurements to confirm that indoor concentrations for BTEX were influenced by gasoline-related sources, and to determine if pollutants originate from vapor or exhaust emissions. Furthermore, we compare the RIOPA concentrations with those from a fixed monitoring site. Last, we estimate weekly cumulative exposure to benzene in homes due to vehicles in attached garages and in cars during heavy traffic, and their respective cancer risks.

METHODOLOGY

This research is based on an analysis of data from a sample of homes without resident smokers that participated in the RIOPA study. Data were made available by the Health Effects Institute [21]. Approximately 100 residences volunteered in each of Los Angeles County, California, Elizabeth, New Jersey, and Houston, Texas. Participants in Houston and Elizabeth constitute a convenience sample, while the participants from Los

Angeles were a subset from a randomly selected sample of individuals from a previous study.

Weisel et al. [22] provide a detailed description of the RIOPA field and measurement protocols. From 1999 to 2001, homes were monitored during two 48-hour periods that were approximately three months apart. MTBE was still in use as a gasoline additive in the three studied cities during the RIOPA study. Air samples were collected concurrently inside and outside of each home. BTEX and MTBE were monitored using Organic Vapor Monitors (OVM 3500, 3M Company, St. Paul, MN, USA). Concentrations at or below the method detection limit (MDL) for a compound were censored by replacement with half the MDL concentrations. In addition to monitoring the air, building characteristics and daily household activity patterns were collected during each of the sampling sessions by means of questionnaires and walkthrough surveys. Air exchange rates (AER) were simultaneously measured using a perfluorocarbon tracer (PFT) method and were reported as time-averaged values for the sampling period.

In the analysis of the RIOPA dataset, values from the first home visit were usually selected for categorical data when the first and second sampling sessions differed. Averages were calculated when indoor (C_{in}) and outdoor (C_{out}) concentrations, and AER were available for the two monitoring sessions, because these are dependent variables that describe a single household.

Constraints reduced the overall sample size of the RIOPA database from 311 to 114. Apartments (n = 108) and single-family attached homes (n = 11) were not included in the evaluation because pollutants from adjacent dwellings can infiltrate through shared walls and affect the measured concentrations. Households where someone smoked during a sampling period (n = 1) or that had gasoline-powered devices other than vehicles

inside the house (n = 16) were excluded to limit the assessment on the effects from parked vehicles. Ventilation rates greater than 5 h^{-1} (n = 2) were also excluded because the PFT method is unreliable at these values. Residences where volumes were recorded to be less than 80 m^3 (n = 3) were not included because it is highly probable that these values were not correct. Houses where information on the location of the parked car was missing or where vehicles were parked in different locations during each sampling period (n = 36) were excluded from the analysis. Only homes with measurements for C_{in}, C_{out} and AER were evaluated. Missing information and other constraints further reduced the dataset size by 20 homes.

Nonparametric statistical analyses were utilized because the data were generally positively skewed. Associations between variables were evaluated with Spearman rank-correlation coefficients (r_s); coefficients were considered statistically significant at $p \leq 0.05$. The Wilcoxon sign-rank test was used to assess differences between paired samples, such as indoor and outdoor concentrations that were concurrently measured. The Wilcoxon rank-sum test was utilized to evaluate differences between two independent samples, such as indoor concentrations from homes with vehicles parked in an attached garage and homes with vehicles in an adjacent carport. Similarly, the Kruskal-Wallis test was used with three or more levels. Differences were considered statistically significant at $p \leq 0.05$. SPSS (version 15.0, SPSS Inc.) was employed for these analyses.

RESULTS

The majority of the residences included in this analysis were located in Houston (HO; n = 55), followed by Los Angeles (LA; n = 38) and Elizabeth (EL; n = 21). These houses were either single-family detached (SFD) structures (n = 99) or manufactured homes (n = 15). Values of C_{in} and C_{out} for BTEX and MTBE in these three cities are

summarized in Table 1. Toluene had the lowest percentage of indoor (61%) and outdoor (34%) concentrations greater than the MDL, likely because of high toluene background levels in the charcoal pads of the OVMs [23]. At least 79% of the indoor concentrations and 47% of the outdoor concentrations for the remaining compounds were above their respective MDLs, with the exception of indoor ethylbenzene concentrations in Elizabeth (63%).

Table 1. Indoor and outdoor BTEX and MTBE concentrations ($\mu g/m^3$) by city.

Compound	Indoor				Outdoor				Indoor vs. Outdoor[a]
	Mean	SD	Median	% > MDL	Mean	SD	Median	% > MDL	
***Los Angeles, CA* (*n* = 38)**									
Benzene	2.14	1.12	2.20	93	2.25	1.39	1.98	83	
Toluene	11.2	7.04	9.48	61	8.94	6.31	6.76	39	I*
Ethylbenzene	1.92	2.18	1.21	88	1.39	0.75	1.39	85	
m&p-Xylene	5.67	7.26	3.94	95	4.08	2.69	3.87	98	
o-Xylene	1.97	2.12	1.58	93	1.57	0.92	1.48	90	
MTBE	7.81	4.97	6.52	97	9.19	5.71	7.12	98	O*
***Elizabeth, NJ* (*n* = 21)**									
Benzene	1.59	1.13	1.40	88	1.23	0.65	1.21	50	I*
Toluene	11.7	9.69	7.55	71	6.83	5.39	3.02	34	I**
Ethylbenzene	2.11	3.55	1.07	63	1.13	0.82	1.02	47	
m&p-Xylene	5.93	11.0	3.99	92	2.39	1.33	2.30	100	I**
o-Xylene	1.85	2.56	1.14	87	0.88	0.42	0.98	82	I**
MTBE	4.84	4.23	3.50	79	4.49	4.47	3.91	89	
***Houston, TX* (*n* = 55)**									
Benzene	5.45	4.77	3.64	100	2.86	2.39	2.29	100	I**
Toluene	17.4	22.7	10.4	72	5.51	3.51	4.51	43	I**
Ethylbenzene	2.72	3.50	1.82	100	1.01	0.75	0.90	94	I**
m&p-Xylene	7.87	12.3	5.10	100	2.84	1.98	2.41	99	I**
o-Xylene	2.70	4.12	1.86	98	1.05	0.68	0.96	93	I**
MTBE	15.5	23.7	6.88	100	10.4	17.1	5.28	96	I*

Abbreviations: MDL, method detection limit; MTBE, methyl *tert*-butyl ether.
[a]I: indoor concentrations were statistically higher than outdoor concentrations; O: outdoor concentrations were statistically higher than indoor concentrations.
*$0.01 < p \leq 0.05$, **$p \leq 0.01$

In each of the studied cities, correlations between indoor MTBE and indoor BTEX concentrations ($0.45 \leq r_s \leq 0.65$) were statistically significant, with the exception of MTBE and toluene in LA ($p = 0.07$). These correlations indicate that C_{in} for BTEX partly originated from gasoline-related sources because MTBE is a tracer for this fuel.

Statistical comparisons between C_{in} and C_{out} were used to examine source location. Table 1 indicates that in the Houston homes C_{in} was statistically higher than C_{out} for all VOCs. This suggests that sources were within or close to the living area, which was the case for 93% of the households that reported having a parked vehicle nearby during the study. Residences in Elizabeth had indoor and outdoor MTBE concentrations that were not statistically different; only 5% of these homes had cars. However, C_{in} for benzene, toluene and the xylenes were statistically higher than C_{out}, which implies that indoor sources for these VOCs were dominant over ambient mobile sources. In California, C_{in} and C_{out} were statistically similar for all compounds but MTBE ($C_{out} > C_{in}$, $p \leq 0.05$) and toluene ($C_{in} > C_{out}$, $p \leq 0.05$). This indicates that outdoor gasoline-related sources were driving indoor concentrations for every contaminant but toluene, even though 47% of the residences had a vehicle next to the occupied space. Differences among cities in terms of the percentage of homes that had parked cars near the living area during the sampling period may explain why Houston generally had the highest median C_{in} values for all VOCs, whereas Elizabeth usually had the lowest concentrations.

Variations in ventilation rates (Table 2) also likely contributed to differences in indoor BTEX and MTBE concentrations among cities. Low AERs in Houston (median = 0.48 h^{-1}) limited the dilution of contaminants generated close or within the occupied space with fresh air, whereas high AERs in Los Angeles and Elizabeth (median = 1.1 h^{-1} for both cities) increased the contribution from ambient sources to C_{in}.

Table 2. Air exchange rates (h^{-1}) by city, and by building type and location of parked car.

Scenario	*n*	AER (h^{-1})		
		Mean	SD	Median
City				
Los Angeles, CA	38	1.45	1.11	1.06
Elizabeth, NJ	21	1.49	1.21	1.06
Houston, TX	55	0.68	0.60	0.48
Studied scenarios				
1: SFD home, car in attached garage	14	0.48	0.25	0.50
2: SFD home, car in detached garage	7	0.81	0.29	0.65
3: SFD home, car in carport	34	0.82	0.65	0.58
4: Manufactured home, car in carport	15	1.14	0.91	0.77
5: SFD home, no car in attached garage	8	1.30	1.36	0.71
6: SFD home, no car and no attached garage	36	1.56	1.27	1.03

Abbreviations: AER, air exchange rate; SFD, single-family detached.

Discrepancies in AER were likely affected by how households conditioned the indoor space. The mean (median) percent of the sampling period in which households reported to have conditioned the air (P_{cond}) was 50% (50%) in Houston homes, while much lower values of 4.4% (0%) and 7.6% (0%) were observed in LA and EL, respectively. Conversely, the percent of monitoring time in which windows were reported to have been open (P_{window}) was much lower in Houston (mean = 11%, median = 0%) than in Elizabeth (mean = 18%, median = 0%) and Los Angeles (mean = 39%, median = 26%). Statistically significant correlations were observed between AER and P_{cond} in Houston (r_s = -0.27), and between AER and P_{window} in LA (r_s = 0.62) and HO (r_s = 0.39).

The influence of parked cars and ventilation rates on indoor concentrations of BTEX and MTBE was further evaluated. Because of the small sample sizes, data from LA, EL and HO were combined. Residences with vehicles next to the living area during the sampling period (n = 70) had indoor concentrations that were statistically higher than in homes without such sources (n = 44) for all VOCs but toluene and m&p-xylene. The

ratio of median C_{in} values in homes with and without cars ranged from 1.1 (m&p-xylene) to 2.0 (benzene).

The increase in indoor concentrations due to indoor sources was estimated by subtracting C_{out} from C_{in} (ΔC). This increase was statistically higher in homes with cars than in residences without automobiles for all pollutants but MTBE ($p = 0.12$); variations in the proximity of parked vehicles may have influenced the MTBE results. Median ΔC values ranged from -0.01 $\mu g/m^3$ (MTBE) to 4.7 $\mu g/m^3$ (toluene) when cars were present, and from -0.37 $\mu g/m^3$ (MTBE) to 0.71 $\mu g/m^3$ (toluene) in homes without automobiles. Ventilation rates in homes with vehicles (median = 0.59 h^{-1}) were statistically lower than in residences without vehicles (median = 1.0 h^{-1}).

The effect of source proximity was investigated by examining the six scenarios illustrated in Figure 1: single-family detached (SFD) homes with cars in the attached garage (Scenario 1; $n = 14$), detached garage (Scenario 2; $n = 7$), or adjacent carport (Scenario 3; $n = 34$); manufactured homes with cars in adjacent carports (Scenario 4; $n = 15$); SFD homes with attached garages but no cars (Scenario 5; $n = 8$); and SFD homes without both attached garages and cars (Scenario 6; $n = 36$). Residences in scenarios 3 and 4 were not combined because their indoor concentrations were statistically different. In general, C_{in} for BTEX compounds and MTBE were statistically significantly correlated ($0.34 \leq r_s \leq 0.86$) in all of the studied scenarios, but in cases 4 and 5 where indoor BTEX concentrations appear to have been dominated by non-gasoline-related sources. Single-family detached homes with cars in attached garages were the only case where C_{in} was statistically higher than C_{out} for all pollutants. The SFD homes with cars in attached garages or carports had the highest median C_{in} for all compounds. In contrast, households without both attached garages and vehicles had the lowest median C_{in} values for all VOCs but toluene. Indoor concentrations in homes with cars in attached garages

were likely negatively affected by low house ventilation rates (median = 0.5 h^{-1}) as indicated in Table 2. Residences with vehicles in detached garages were the only case where C_{in} and C_{out} were statistically similar for all contaminants, although the small sample size (n = 7) may have influenced the inability to detect a statistical difference.

Increase in indoor concentrations (relative to outdoors) for all compounds and studied scenarios are shown in Figure 2. Single-family detached homes with vehicles in attached garages had the highest median ΔC for benzene (1.2 $\mu g/m^3$), toluene (6.4 $\mu g/m^3$), m&p-xylene (2.6 $\mu g/m^3$) and MTBE (2.7 $\mu g/m^3$), and relatively large values for ethylbenzene (0.69 $\mu g/m^3$) and o-xylene (0.91 $\mu g/m^3$). Additionally, these homes also had the highest median indoor to outdoor concentration ratios (C_{in}/C_{out}): benzene = 2.0, toluene = 2.7, ethylbenzene = 2.1, m&p-xylene = 2.1, o-xylene = 3.3, MTBE = 1.4. The SFD residences with automobiles in carports tended to have the second highest median ΔCs for all VOCs. For the remaining scenarios, excluding homes with detached garages, median ΔC values were greater than zero for BTEX but not MTBE. Residences with detached garages had median ΔC values that were less than zero ($C_{in} < C_{out}$) for all compounds but toluene. Furthermore, these houses had the lowest median C_{in}/C_{out} ratios for BTEX (0.89 to 1.0), and the same median ratios as homes without cars for MTBE (0.92).

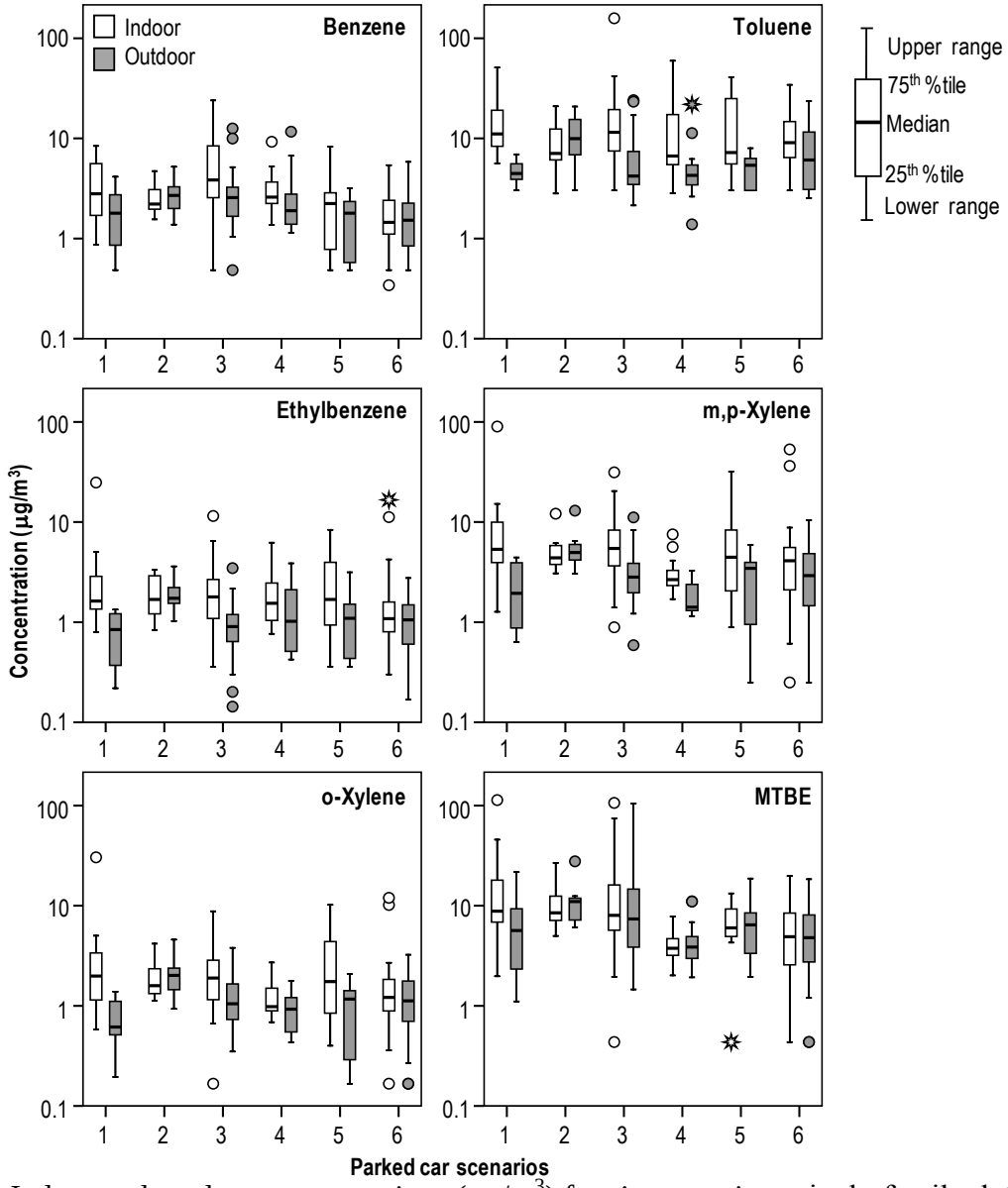

Figure 1. Indoor and outdoor concentrations (µg/m^3) for six scenarios: single-family detached (SFD) homes with cars in the (Scenario 1; *n* =14) attached garage, (Scenario 2; *n* = 7) detached garage, or (Scenario 3; *n* = 34) adjacent carport; (Scenario 4; *n* = 15) manufactured homes with cars in adjacent carports; (Scenario 5; *n* = 8) SFD homes with attached garages but no cars; and (Scenario 6; *n* = 36) SFD homes without both attached garages and cars. 'o' and '∗' indicate values between 1.5 and 3, and > 3 box lengths, respectively, from the 25th or 75th percentiles.

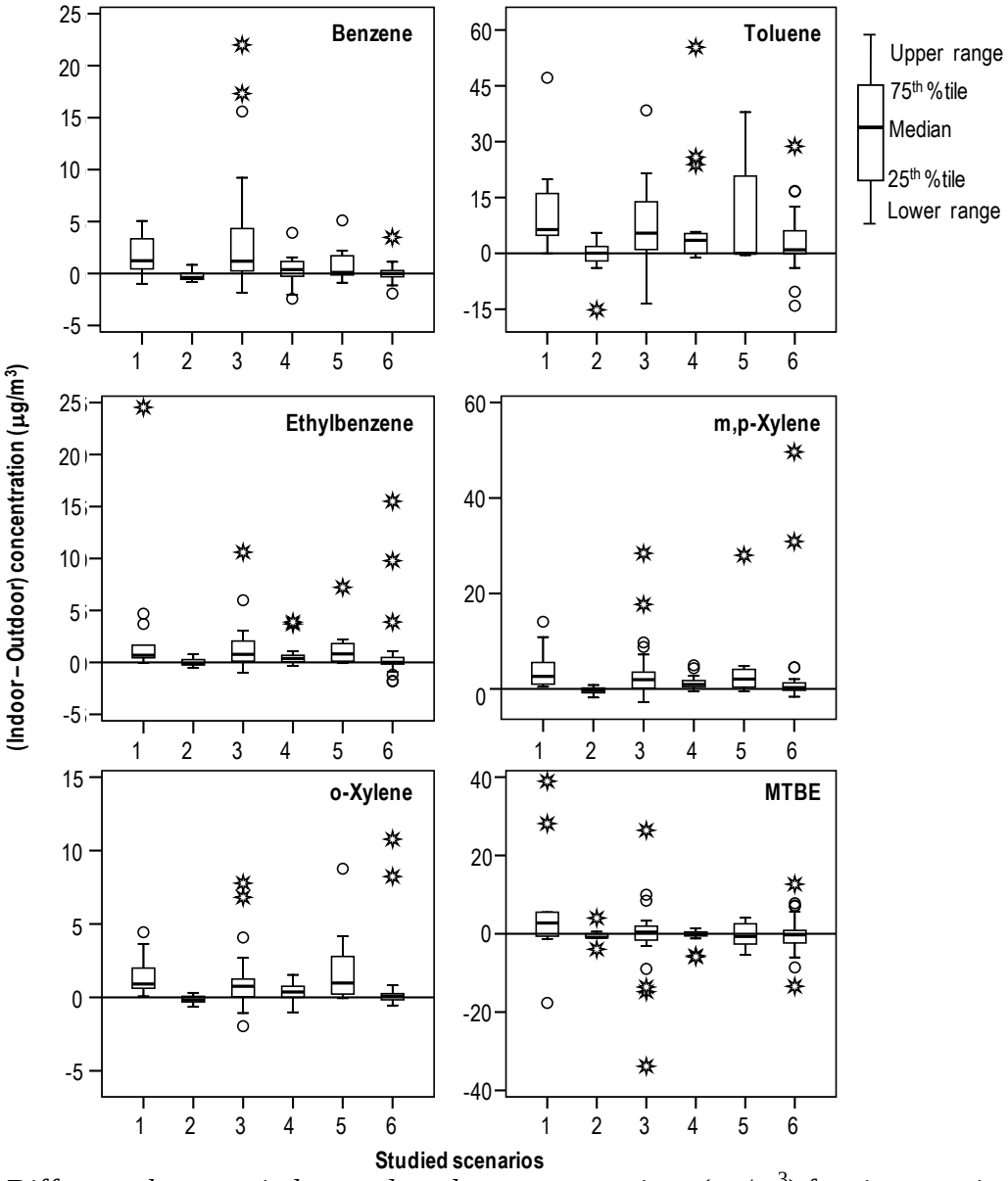

Figure 2. Difference between indoor and outdoor concentrations (µg/m^3) for six scenarios: single-family detached (SFD) homes with cars in the (Scenario 1; *n* =14) attached garage[a], (Scenario 2; *n* = 7) detached garage, or (Scenario 3; *n* = 34) adjacent carport[b]; (Scenario 4; *n* = 15) manufactured homes with cars in adjacent carports; (Scenario 5; *n* = 8) SFD homes with attached garages but no cars; and (Scenario 6; *n* = 36) SFD homes without both attached garages and cars. 'o' and '∗' indicate values between 1.5 and 3, and > 3 times the interquartile range, respectively, from the 25th or 75th percentiles. [a]m&p-xylene = 89 µg/m^3, o-xylene = 30 µg/m^3 and MTBE = 109 µg/m^3 were not included for clarity. [b]Toluene = 155 µg/m^3 and MTBE = 103 µg/m^3 were not included for clarity.

The percent contribution of indoor sources to indoor concentrations (I_{cont}) was calculated by dividing ΔC by C_{in}. This contribution was assumed to be zero in homes where ΔC was negative (i.e., $C_{out} > C_{in}$). Figure 3 shows the cumulative distribution curve for I_{cont} for four of the six studied scenarios; cases 2 and 5 were omitted because of their small sample size. In general, I_{cont} for most compounds was the highest in residences with automobiles in attached garages; median values ranged from 30% (MTBE) to 52% (ethylbenzene). The SFD homes and manufactured homes with cars in carports had the next highest I_{cont}. Their median values were relatively similar, varying from 5% (MTBE) to 51% (ethylbenzene), and from 0% (MTBE) to 50% (ethylbenzene), respectively. Homes without both attached garages and cars typically had the lowest indoor contributions; the median ranged from 0% (MTBE) to 34% (toluene).

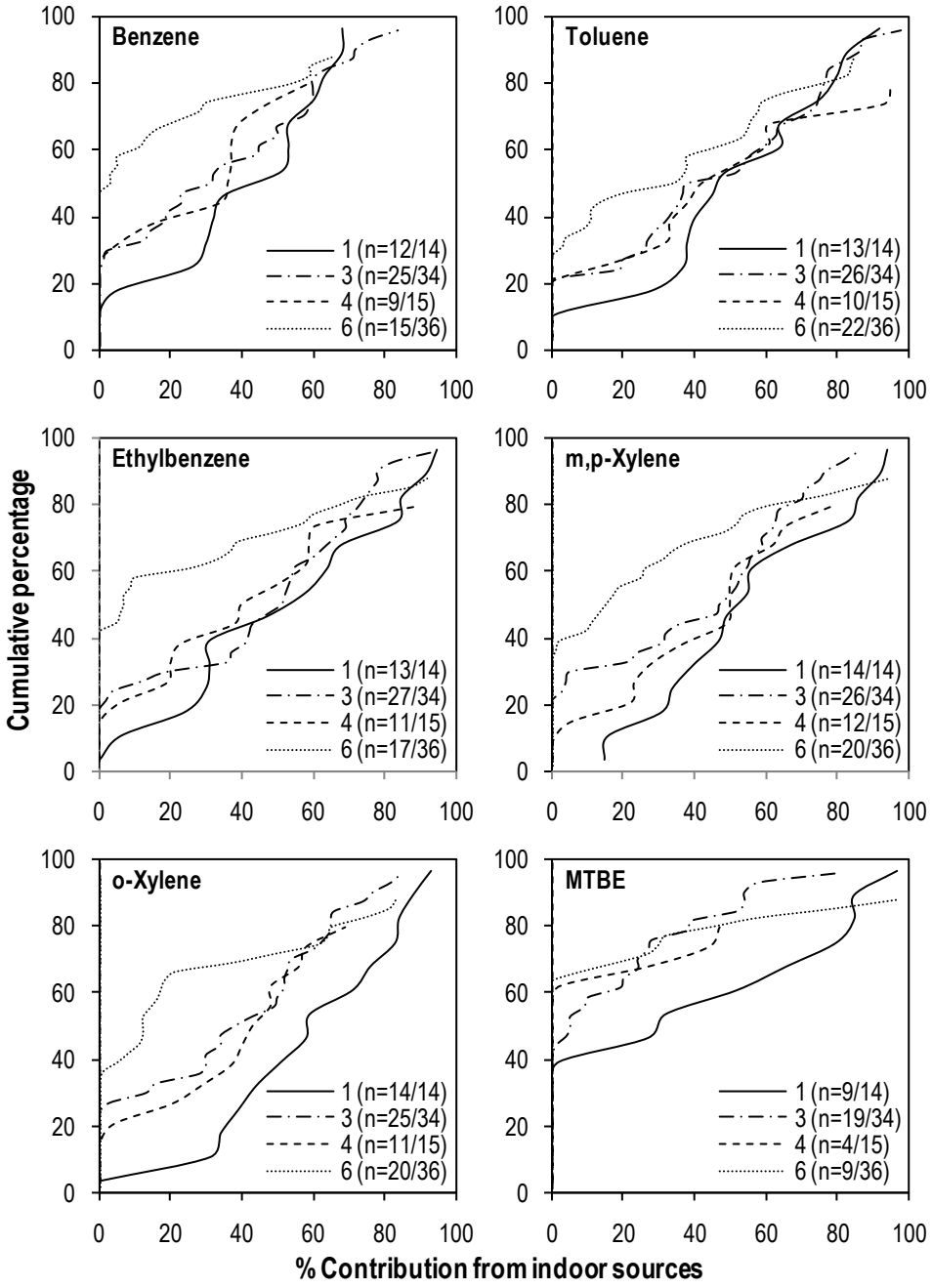

Figure 3. Cumulative distribution functions for the percent contribution from indoor sources to indoor concentrations for four scenarios (n^a): single-family detached (SFD) homes with cars in the (Scenario 1) attached garage, or (Scenario 3) adjacent carport, (Scenario 4) manufactured homes with cars in adjacent carports, and (Scenario 6) SFD homes without both attached garages and cars. Cases 2 and 5 were excluded because of small sample size. [a]Number of homes where indoor concentrations were higher than outdoor concentrations/total number of homes.

DISCUSSION

Results from the RIOPA investigation and those of others [9, 24] indicate that BTEX is nearly ubiquitous indoors. In the RIOPA homes, these pollutants partly originated from gasoline-related sources given that MTBE, a VOC emitted almost exclusively by gasoline, was concurrently detected indoors. Outdoor sources in urban areas contribute considerably to background concentrations of BTEX and MTBE; however, parked vehicles adjacent to residential living areas can exacerbate C_{in}. Batterman et al. [10], Dodson et al. [9], and Thomas et al. [11] monitored single-family homes and determined that BTEX and MTBE concentrations in attached garages can exceed C_{in} by an order of magnitude. Batterman et al. [10] estimated the median contribution from sources in the garage to indoor BTEX concentrations to range from 47% (toluene) to 65% (benzene) using field measurements and multi-zonal mass-balance models to approximate the airflow between these two areas. Dodson et al. [9] reported comparable median garage contributions (i.e., 30% for toluene to 44% for m&p-xylene) after following a similar procedure. Our results were also comparable, with I_{cont} ranging from 43% (benzene) to 58% (o-xylene) in SFD homes with vehicles in attached garages, even though our estimates do not distinguish between emissions within the living area or the garage. With regard to MTBE, our estimates for I_{cont} (median = 30%) were similar to those from Dodson et al. [9] (median = 32%). Variations that affected these results include the amount of air that infiltrated from the garage into the living space, house air exchange rates, and source strengths within the garage, living area and outdoors.

In addition to being emitted by mobile sources, ethylbenzene and xylene isomers tend to be concurrently emitted by paint-related products [13]. This may explain the high statistically significant correlations among indoor concentrations for these VOCs that we noted in homes without vehicles ($r_s > 0.79$), and that Jia et al. [25] observed in homes for

the population at large ($r_s > 0.92$). Since toluene is found in a wider variety of consumer products, such as cleaners, paints, polishes and adhesives [13, 14], homes without cars had indoor concentrations for toluene that had lower correlation coefficients with the other BTEX compounds ($r_s \cong 0.45$). However, these results may have also been affected by the low percentage of toluene measurements above the MDL.

We used the ratio of MTBE to benzene indoor concentrations to examine if these compounds originated from gasoline vapors or car exhaust. Low MTBE/benzene ratios indicate that tailpipe emissions are dominant because during combustion the amount of MTBE decreases while benzene is enriched due to toluene and xylene dealkylation [12]. Alternatively, high MTBE/benzene ratios suggest a significant contribution from evaporative emissions from hot soak, fuel tank "breathing" due to diurnal temperature and barometric changes, and/or fuel system leakage. The Desert Research Institute (DRI) [12] reported ratios for various microenvironments in Houston (freeway = 1.7 – 2.9, in-cabin underground garage = 2.4 – 3.2, in-cabin refueling = 25 – 42, and outdoor refueling = 29 – 56), car exhaust (Houston = 0.44 – 1.4, Los Angeles = 0.43 – 1.1), and liquid gasoline (Houston = 13 – 18, Los Angeles = 12 – 20).

Our estimates for MTBE/benzene ratios are shown in Figure 4; we excluded nine houses that had indoor benzene measurements that were both below the MDL and less than 1 $\mu g/m^3$. Vehicle exhaust appeared to drive C_{in} in about half of the homes given that median ratios for the six studied scenarios ranged from 1.5 to 4.2. For most of the homes with ratios above the median, a mixture of tailpipe and gasoline vapor emissions seemed to have influenced indoor concentrations of gasoline-related VOCs because the six scenarios had 80^{th} percentile ratios that did not exceed 7. Evaporative emissions were substantial in four households where MTBE/benzene values were greater than 11. Two of these were SFD homes with cars in the attached garage. The other two residences did

not have cars next to the living area; we speculate that these participants failed to report the presence of indoor gasoline sources given that indoor MTBE concentrations were greater than C_{out} by at least 7.8 μg/m^3.

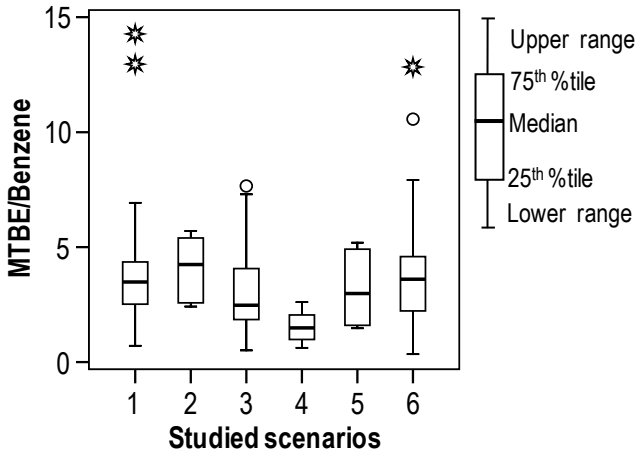

Figure 4. Ratio of MTBE to benzene indoor concentrations for six scenarios: single-family detached (SFD) homes with cars in the (Scenario 1; *n* =14) attached garage, (Scenario 2; *n* = 7) detached garage, or (Scenario 3; *n* = 33) adjacent carport; (Scenario 4; *n* = 15) manufactured homes with cars in carports; (Scenario 5; *n* = 6) SFD homes with attached garages but no cars; and (Scenario 6; *n* = 30) SFD homes without both attahced garages and cars. Twenty homes were excluded where indoor benzene concentrations were both lower than the MDL and less than 1 μg/m^3. 'o' and '∗' indicate values between 1.5 and 3, and > 3 times the interquartile range, respectively, from the 25th or 75th percentiles.

Since air pollutants emitted by mobile sources are commonly assessed by governmental agencies, we examined how measurements from RIOPA-TX compared to those from monitoring stations. We obtained ambient concentrations for these compounds from the Texas Commission on Environmental Quality (TCEQ), which operated one monitoring station near the sampled homes using canisters and reported time-averaged 24-hour concentrations that were collected every six days [26]. We paired data from RIOPA with the TCEQ concentration measured the week before the home was

sampled. Outdoor concentrations for ethylbenzene, toluene and m&p-xylene from RIOPA were statistically higher than those reported by TCEQ, probably because of sources near the residences that were not detected by the fixed monitoring site or because of differences in meteorology between the sample sites [27]. Only outdoor o-xylene concentrations from TCEQ were statistically higher than those from RIOPA. Indoor BTEX and MTBE concentrations from the RIOPA homes were statistically higher than ambient levels from TCEQ. For benzene and MTBE, residences with cars in attached garages (n = 10) had median indoor concentrations (benzene = 3.8, MTBE = 11 $\mu g/m^3$) that showed the highest discrepancy from their respective median TCEQ values (benzene = 2.4, MTBE = 6.3 $\mu g/m^3$).

To place some of our results into context, we used the RIOPA data to estimate weekly cumulative exposure to benzene in two microenvironments in Houston: homes with vehicles parked in attached garages and cars driven in a freeway with heavy traffic during commute to and from work. We selected Houston because 71% of the RIOPA homes that correspond to the first microenvironment were located in this city. We calculated exposure by multiplying concentration by the exposure time and dividing the result by the total time spent in the two microenvironments. For homes, we used the mean ΔC for benzene (2.3 $\mu g/m^3$) to better evaluate the effect of parked vehicles and assumed that individuals spend 70% of the week in their house [20]. For cars, we assumed a mean in-cabin concentration of 6.1 $\mu g/m^3$ [12], an average commute time to work of 26 minutes [28], a mean travel time from work equal to the commute time to work, and a five-day work week. Weekly exposure to benzene was 2.2 $\mu g/m^3$ in homes with cars in attached garages, and 0.22 $\mu g/m^3$ in cars during commute to and from work. These results indicate that even though increases in indoor concentrations due to vehicles in attached garages are relatively small, the fact that we spend a large amount of time in

our homes can lead to exposures to benzene that are ten times higher than what we may experience in more severe microenvironments that we typically frequent such as heavily congested highways. Additionally, these increases in benzene concentration due to vehicles in garages could lead to a mean cancer risk of 17 per million population in Houston, based on an inhalation unit risk factor of 7.8×10^{-6} $m^3/\mu g$ [8]. The EPA benchmark for exposure to potential carcinogens is 1 per million.

Methods to reduce indoor residential concentrations of VOCs emitted by parked vehicles next to the living quarters need special attention because 55% of single-family homes and manufactured homes in the U.S. have an attached garage or carport [29]. ASHRAE Standard 62.2 [30] describes measures to prevent the migration of pollutants from attached garages into the occupied area in new housing, although these are also applicable to existing residences. These recommendations include (1) sealing vertical and horizontal surfaces shared by these two spaces; (2) avoiding placement of HVAC components in the garage; (3) limiting the total air leakage of HVAC components, especially when located in the garage; and (4) maintaining the living area at a higher pressure than that of the garage. It is not evident that carports are a good alternative to attached garages; SFD homes with cars in carports had relatively high median ΔC values (e.g., benzene = 1.2 $\mu g/m^3$, MTBE = 0.42 $\mu g/m^3$). Conversely, manufactured homes with vehicles in carports had much lower median ΔC values (e.g., benzene = 0.38 $\mu g/m^3$, MTBE = -0.10 $\mu g/m^3$). Various factors could have affected these results such as the location of windows and doors with respect to the carport, the number of parked cars, and meteorological conditions. Infiltration of pollutants into the living quarters can be limited by tightening the house envelope, specifically close to the carport. In addition to the measures just described, the design of new residences could be improved by incorporating detached garages. Our results indicate that homes with detached garages

had minimal increases in indoor concentrations of BTEX and MTBE, which suggests that in addition to cars, non-gasoline-related sources for BTEX may have been stored in the detached garage where they were not as likely to affect the occupied space.

CONCLUSION

Our evaluation of the RIOPA database supports prior work on the detrimental effects of attached garages on indoor air quality in residences, and provides insight on how variations in the proximity of parked vehicles to the living area affect indoor concentrations of BTEX and MTBE. Results from our assessment of six parking scenarios indicate that homes with attached garages were affected the most by cars. The percent contribution of indoor sources to C_{in} in these residences ranged from 30 to 58%. Moreover, houses with attached garages generally had the highest median increases in indoor concentrations (relative to outdoor concentrations) for BTEX compounds ($0.69 \leq \Delta C \leq 6.4$ $\mu g/m^3$), a trend that was likely affected by lower ventilation rates for homes with attached garages. While the ΔC values may appear inconsequential, increases in indoor benzene concentrations can lead to weekly cumulative exposures that are ten times higher than those experienced while commuting in a car in heavy traffic, and to mean excess cancer estimates that are 17 times higher than the EPA benchmark. Strategies to reduce exposure to gasoline-related VOCs in homes include sealing surfaces shared by the living quarters and the garage, and not placing components of the air conditioning system in the garage. Furthermore, our results suggest that improving the design of homes by incorporating detached garages could be an alternative solution.

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Vita

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